South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde
P.O. Box 643, Cape Town Posbus 643, Kaapstad

Cape Town, 15 March 1958 Vol. 32 No. 11 Kaapstad, 15 Maart 1958
Weekly 2s. 6d. Weekliks 2s. 6d.

SOME OBSERVATIONS ON ANAL ABSCESS AND FISTULA

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At the beginning of the 16th century John Arderne developed a treatment of fistula-in-ano which was to last for over 450 years and is still used today. His practice was to lay open the track, and then to dress the wound without cauterization (which was at that time considered essential to prevent infection). Whenever possible he lived in the patient's home to tend the dressings personally until healing was complete; from all accounts his results compared favourably with those of contemporary surgeons.

The next major advance in the understanding of anal abscess and fistula came in 1880, when Desfosses and Herrmann[1] described the various types of glands found in the ano-rectal region. For clarity and accuracy their treatise is still unsurpassed today. They recognized *inter alia* the 'deep intramuscular' anal glands—long canals or ducts opening into the valves of Morgagni, whose outer ramifications penetrate the internal sphincter and may even enter the external sphincter. They believed these glands to be morphologically equivalent to the cloacal sex glands of dogs. They also suggested that infection of these glands might account for abscess formation and the subsequent development of fistula.

Both Miles[2] and, later, Lockhart-Mummery[3] in their text-books on rectal disease stated unequivocally that fistula-in-ano was a complication following on abscess, yet this does not appear to be common knowledge even today. Kratzer and Dockerty[4] demonstrated the intramuscular glands in human embryos by means of serial sections; each embryo had about 3-5 glands, of which 2-3 were posterior (Fig. 1). It was also shown that ducts ran caudad in most instances. The proximal lining was squamous and the alveolar cells were columnar. About 30% of the specimens gave a positive mucin stain. Professor Harris[5] has shown that these deep intramuscular anal glands develop before the muscularis mucosae (which in the anus we prefer to call the muscularis submucosae) and penetrate at a stage when internal and external sphincters have not yet overlapped. Several authors[6, 7] have demonstrated the presence of epithelium in the track of excised fistulae, strongly suggesting a glandular origin.

One must therefore conclude that ischio-anal abscess (a more correct anatomical term than 'ischio-rectal') and fistula-in-ano are merely two stages in the same disease process. Goodsall[2] was probably not quite correct in stating that posterior fistulae all open in the mid-line; nor was Hiller[8] right in supposing that suppuration in the ischio-anal space follows along the peri-vascular plane to the anal epithelium. The cloacal sex glands of other mammals are usually paired in two groups opening into the posterior anus on either side of the mid-line. This seems a reasonable explanation of the facts as we know them; added support is given by a study of these glands in the gorilla, in whom (as in the human) they are vestigial.

It will be clear that incision only of an ischio-anal abscess will lead to a high incidence of recurrence. It is usually possible at operation to demonstrate the internal opening, and this should be laid open and the lining excised or curetted. If this cannot be done, the patient should be warned that a second staged operation may be necessary. In dealing with fistula it should also be remembered that glandular tissue may line portions of the track, especially if there has not been any acute or gross infection. If the fistula is only laid open, these glandular cell-nests may form the nidus for later troubles. It would seem logical to *excise* fistulae; at the same time removing scar tissue which otherwise delays healing.

The occasional rectal surgeon is often concerned about how much of the sphincter muscles he may safely divide without producing incontinence. As a general rule, if there is any doubt whatever in the surgeon's mind, he should play safe, open the lower portion of the track, and place a thick braided silk ligature round the compromised muscle. This promotes fibrous tissue reaction and allows the muscle to be divided a week or so later without danger of retraction. In fact one can divide much more muscle than most physicians would believe possible. To understand the reason for this, a few words must be said about sphincteric function: It has been assumed in the past that a sphincter acts by virtue of its pinch-cock or purse-string effect. Detailed study of several body sphincters has shown that this is not correct, and that a combination of angulation plus valve-flap effect is more

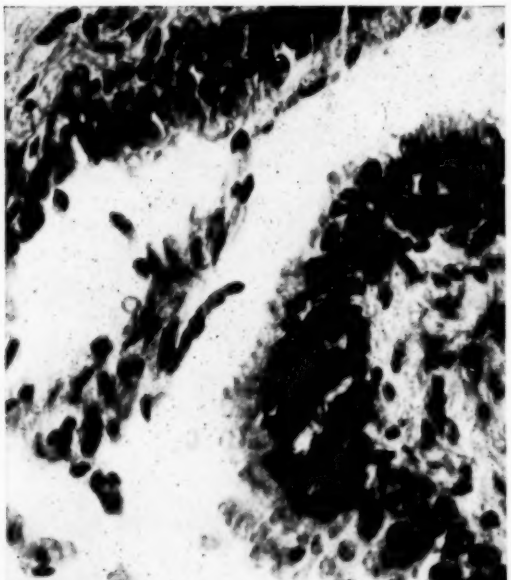

Fig. 1. Microscopic section taken through the duct of an anal intramuscular gland, ×200. Note the columnar secretory mucosa merging into cuboidal ductal epithelium near the orifice.

important (e.g. oesophago-gastric and ileo-caecal areas). Our new concept of anal control is that continence depends upon the angle between rectum and anus produced by the pubo-rectalis sling of the levator ani. Provided this remains anatomically and functionally intact, the remainder of the internal *and* external sphincters may be divided. Fibres from the levator ani blend with those of the external sphincter, and these two must be regarded as a single physiological unit. The three divisions of Milligan and Morgan are artificial and have, I believe, outlived their usefulness.[9]

The rationale of a wide accompanying skin excision is to allow adequate external drainage until the slower-growing internal mucosal wound has healed, and to have a flat smooth wound without danger of overlap or pocket formation. It is most important that the surgeon should attend these wounds personally; and in the more serious cases we review the wound under general anaesthesia in the operating theatre every 7-10 days. Pockets are opened and overhanging skin or mucosal edges are trimmed. We have found this procedure invaluable.

As long ago as 1927[10] Gabriel raised the question of skin grafting of very large wounds, and this is without doubt a useful procedure in selected cases. Small 'postage-stamp' Thiersh grafts are used. They should not be sutured, but are kept in position by means of a firm elastoplast dressing. The bowels should be confined for 5-6 days and antibiotic cover should be used.

OPSOMMING

Ischio-anal-abses en buis is twee betoginge van dieselfde siekte. In die meeste gevalle is abses 'n gevolg van aansteking van die ‚diep intramuskulaar' anus-kliere; en 'n buis volg altyd 'n abses. As die chirurg 'n abses insny, moet hy ywerig vir die innerlike opening soek, en dit ook oopsny. Dit is beter om 'n buis *uit* te sny as net oop te sny. Ons glo vandag dat die analsirkelspier nie deur middel van sy knypaksie werk nie, maar op die slinger van die pubo-rektalis afhang om onmatigheid te voorkom. Die chirurg kan dus die grootste deel van die spier veilig deursny; maar as hy nie seker daarvan is nie, is dit beter om die buis in twee stadiums te opereer. 'n Oorplant van vel kan in uitgekieste gevalle nuttig wees.

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EDITORIAL

BONE DISEASE IN RENAL FAILURE

It looks as though we may have to re-orientate our ideas concerning the type of bone disease produced by renal glomerular azotaemic failure. We have long been taught that 'renal rickets' was the wrong name to apply to the metaphyseal lesion seen in childhood renal failure—that the bone disease was osteitis fibrosa, caused either by the acidosis or by a secondary hyperparathyroidism.

At a meeting of the Renal Association,[1] both Stanbury and Dent agreed that, in fact, florid rickets and osteomalacia sometimes occur in renal failure, and further that they may appear with or without coincident osteitis fibrosa. Stanbury showed histological preparations of the metaphyses from uraemic children which were typical of true rickets—by definition, 'a failure of mineral deposition to keep pace with the endochondral growth of bone'. Both authorities agreed that the proximate cause of the osteomalacia was a defective intestinal absorption of calcium, rather than a wastage of minerals in the urine. In comparison with vitamin-D resistant rickets (or osteomalacia) this defect could be overcome by large doses of either calciferol or dihydrotachysterol (AT 10), with radiological evidence of quite rapid healing. There appears to be an acquired insensitivity to vitamin D to account for the metabolic defect, which cannot be blamed on the acidosis or the high serum phosphate. It is possible that some anti-vitamin-D factor circulates in renal failure, but little is known about this.

The osseous changes in the osteitis fibrosa of renal failure are indistinguishable from those of primary hyperparathyroidism. The earliest clinical changes may be seen in radiographs of the fingers, as erosions or 'scalloping' of the cortical surface of the phalanges. It seems extremely likely that a secondary hyperparathyroidism accounts for the osteitis, but the cause of this parathyroid change is not known with any certainty. Stanbury pointed out that rickets or osteomalacia was not a necessary precursor of the hyperparathyroidism. The Albright concept of 'a tendency to low serum calcium' acting as a stimulus to the parathyroids is not proven. As Stanbury further points out, even the development of a hypercalcaemia does not necessarily cause the osteitis to regress. Usually, however, the hyperparathyroidism as well as the osteomalacic changes heal rapidly on large doses of calciferol.

VAN DIE REDAKSIE

BEENSIEKTES BY NIERVERSAKING

Dit lyk asof ons ons idees van die soort beensiektes wat veroorsaak word deur asotemiese versaking van die nierbuisies ietwat moet verander. Dit word ons al lank voorgehou dat dit verkeerd is om die metafise-letsel van nierversaking by kinders ‚nier-rachitis' te noem—dat die beensiekte eintlik fibreuse beenverweking is wat veroorsaak word deur die suurvergiftiging òf deur 'n bykomende oormatige byskildklierwerking.

By 'n vergadering van die *Renal Association*[1] het beide Stanbury en Dent saamgestem dat vol-ontwikkelde rachitis en osteomalasie wel soms in aansluiting met nierversaking voorkom, en dat hulle ook met of sonder samevallende osteitis fibrosa kan voorkom. Stanbury het histologiese preparate van die metafises van ureniese kinders gedemonstreer wat kenmerkend was van egte rachitis—omskryfbaar as ‚'n kondisie waarby die minerale neerslag nie tred hou met die kraakbeengroei (van die been) nie.'

Hierdie twee gesaghebbendes stem saam dat 'n gebrekkige kalsiumopname uit die derm as die onmiddellike oorsaak van osteomalasie beskou moet word, eerder as 'n minerale-verlies in die urien. In teenstelling met rachitis vitamien-D-bestand (of osteomalasie) kan hierdie defek reggestel word met groot dosisse kalsiferol òf dihidrotachysterol (AT10), met radiologiese bewys dat die letsels heel spoedig genees. Daar is blykbaar 'n aangewende ongevoeligheid (of weerstand) vir Vitamien D wat die metaboliese defek verklaar—dit kan nie aan die suurvergiftiging of aan die hoë serum-fosfaatgehalte te wyte wees nie. Dit is moontlik dat die een of ander vitamien-D-bestrydende faktor in die bloed sirkuleer by 'n geval van nierversaking, maar ons kennis hieromtrent is maar beperk.

Die beenveranderings by die osteitis fibrosa van nierversaking kan nie onderskei word van dié wat by primêre oormatige byskildklierwerking voorkom nie. Die vroegste kliniese veranderings kan waargeneem word op X-straalplate van die vingers as wegvretings of ‚scalloping' van die skors oppervlaktes van die vingerbeentjies. Dit lyk baie moontlik dat die beenontsteking deur 'n sekondêre oormatige byskildklierwerking veroorsaak word, maar ons het nog geen duidelike kennis van hierdie byskildklierafwyking nie. Stanbury wys daarop dat rachitis of osteomalasie nie noodwendig 'n voorloper is van die oormatige byskildklierwerking nie. Die Albright-teorie dat ‚'n neiging tot 'n lae serumkalsiumgehalte' as prikkel tot die paratiroïede ageer, is nog nie bevestig nie. Soos Stanbury verder aantoon, lei selfs die ontwikkeling van 'n oormaat kalsium in die bloed nie noodwendig na 'n verbetering van die beenontsteking nie. Gewoonlik herstel die sieklike veranderings van oormatige byskildklierwerking sowel as dié van osteomalasie egter baie gou na groot dosisse kalsiferol.

Dent mentioned a third bone lesion which was occasionally seen in renal failure—osteosclerosis. He remarked that all 3 types of bone disease sometimes occur in steatorrhoea, and here also a resistance to the action of vitamin D could be found, even when it was given parentally.

So the old physicians who talked of renal rickets were not wrong after all, but the bone lesions which do occur in renal glomerular failure with azotaemia are certainly more complex than was formerly imagined. As usual, the more we learn about them, the more there seems to be to find out. One good feature from the clinical angle is that the same treatment—calciferol in high dosage—will cure both the typical types of bone disease even when the renal failure is actually progressing.

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Dent maak melding van 'n derde beenletsel wat soms by nierversaking gesien word—osteosklerose. Hy verklaar dat al 3 soorte beensiektes soms by steatorree voorkom, en ook by hierdie kondisie bied die liggaam weerstand teen vitamien D, selfs wanneer dit parentaal toegedien word.

Die ou geneeshere wat van nier-rachitis gepraat het was dus tog reg, maar die beenletsels wat by nierbuisversaking met asotemie voorkom is gewis baie meer ingewikkeld as wat eers vermoed was. Soos gewoonlik, hoe meer 'n mens daarvan te wete kom, hoe meer skyn daar wat nog geleer moet word. Een goeie ding, klinies gesproke, is dat dieselfde behandeling, nl. groot dosisse kalsiferol, albei die gewone soorte beensiektes sal genees, selfs al vererger die nierversaking.

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'THE HISTORY OF MEDICINE IN SOUTH AFRICA'

Several members have stated that they do not agree that this book should be sent to all members who have not informed the publishers that they do not wish to receive it.

It is common practice for learned societies who publish works of importance to give their members an opportunity of receiving preference in obtaining copies. Experience has shown that if members are asked to order a copy in advance, when a limited edition is being printed, numbers of members omit to do so and are subsequently very disappointed when they are unable to have one on their shelves.

The Medical Association has undertaken the production of this work (it has taken nearly ten years to bring it to publication) as a service to its members and to the country. It has made its members a unique offer through the publisher and it is satisfied that members will be pleased with the result.

Finally, when the book is sent out, all who do not wish to keep it will have the opportunity to return it after inspection and within a limited time.

THE CLINICAL PRESENTATION OF POLIOMYELITIS IN THE YOUNG BANTU CHILD

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In 1951 it was reported that among urban Bantu children silent infection with the virus of poliomyelitis took place early in life, but the incidence of paralytic poliomyelitis was relatively low.[1] However, in 1956, in the largest epidemic of clinical poliomyelitis yet seen in the Johannesburg Bantu, 169 cases of acute anterior poliomyelitis were seen in the Paediatric Unit, Baragwanath Hospital, Johannesburg, in the first half of the year.

Early diagnosis of this disease to prevent admissions to the general wards was considered important, and this paper gives an account of the clinical presentation and the difficulties encountered in diagnosis.

Material

The Paediatric Unit at Baragwanath Hospital serves non-European children under 9 years of age. It is a large unit where 5,839 and 5,996 in-patients, and 73,000 and 88,000 out-patients were seen in 1955 and 1956 respectively. There are no isolation facilities for patients suffering from infectious disease.

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Between January and July 1956 119 cases of poliomyelitis were diagnosed at Baragwanath Hospital, but the records of only 99 could be traced, and these patients are studied in this paper. Of these, 18 were in-patients and the rest were out-patients. In addition, 50 patients with poliomyelitis, diagnosed elsewhere, were referred for physiotherapy, after the acute stage of the disease.

Definitions

The following terms used in this paper are briefly defined:

Non-paralytic poliomyelitis: Evidence of involvement of the central nervous system, with positive cerebrospinal-fluid findings, but without paralysis; with or without recovery of polio virus from the stools. (This was attempted in 3 patients; 2 were positive.)

Encephalitic form of poliomyelitis: Evidence of poliomyelitis, but encephalitic symptoms (coma, stupor or convulsions) dominated the clinical picture.

Bulbar form of poliomyelitis: Cranial-nerve involvement without clouding of consciousness, and with or without involvement of the spinal cord.

Spinal form of poliomyelitis: Paresis or paralysis attributable to involvement of the cervical, thoracic or lumbo-sacral cord.

The spinal cases are further described after the classification of Smith, Harris and Rosenblatt:[2]

Group I	Mild	Minimal isolated muscle weaknesses.
Group II	Moderate	Includes weakness of 2 limbs.
Group III	Moderately severe	Includes flaccid paralysis of one limb with or without isolated muscle weakness elsewhere.
Group IV	Severe	Includes flaccid paralysis of 2 limbs with or without isolated muscle weakness elsewhere.
Group V	Very severe	Includes flaccid paralysis of 3 limbs or respiratory muscle paralysis (diaphragms, intercostals, abdominals).

Presentation

67% of the patients presented with paralysis already present (Table I), although this had not always been recognised as

TABLE I. PRESENTING SIGNS AND SYMPTOMS

Paralysis	66
Fever	11
Cough and fever	7
Pain	5
'Twisted' neck	2
Convulsions	2
Sores in mouth	2
Diarrhoea	1
Nystagmus	1
Swollen fontanelle	1
Listlessness	1
Total	99

such by the mother, who might say, 'Baby won't crawl', or 'Baby has a crooked face'. Only about 1/5th gave a history of preceding feverish illness.

Sex and Age

There was the usual preponderance of boys (Table II),

TABLE II. TABLE SHOWING SEX INCIDENCE

Males	52
Females	36
Sex not recorded	11
Total	99

which has been noticed in most epidemics. The highest age incidence was between 6 and 18 months (36 cases); 3/4ths of the cases were less than 3 years old (Table III).

TABLE III. TABLE SHOWING AGE INCIDENCE

0 to 5 months	2
6 months to 11 months	15
1 year to 17 months	21
18 months to 2 years	13
Over 2 years to 3 years	19
Over 3 years to 4 years	11
Over 4 years to 5 years	6
Over 5 years to 6 years	0
Over 6 years to 7 years	3
Over 7 years to 8 years	2
Age unknown	7
Total	99

Type and Severity

More than 3/4ths of the patients showed the spinal paralytic form of the disease (Table IV); 9% were bulbar, 3% were encephalitic, and only 9% of cases showed the non-paralytic form. When the spinal paralytic cases were grouped according to severity, it was noted that only 31% fell into the mild or moderate category, while 63% were moderately severe, severe or very severe (Table V).

Cerebrospinal Fluid

Lumbar puncture, which was usually an out-patient procedure, was found essential for diagnosis in nearly all cases under 2 years of age. The cerebrospinal fluid findings in 64 of the patients are summarized below.

Of the patients with typical paralytic poliomyelitis, 5 (7·8%) gave normal cerebrospinal fluids. In the other patients the total cell count varied from no cells to 225 cells per c.mm. In half the patients it was less than 60 cells per c.mm. The cells were chiefly lymphocytes, the highest count being 189 per c.mm. In 1/3rd of cases polymorphs were less than 5 per c.mm., and in only 16 patients were there more polymorphs than lymphocytes. Of these, 7 had polymorph counts varying from 79 to 138 per c.mm. (3 were bulbar, 1 polio-encephalitic, and 3 non-paralytic in type). Where the polymorph count was high, only one patient showed a protein value of over 40 mg.%. Where the total cell count was high, the protein was usually, but not invariably, low. Half the patients lumbar punctured showed a protein value below 31 mg.%, and the highest value was 116 mg.%.

TABLE IV. CASES OF POLIOMYELITIS ACCORDING TO AGE AND FORM OF POLIOMYELITIS

Forms of Poliomyelitis	*Unknown age*	0-5 *months*	6-11 *months*	12-17 *months*	18 *mths*-2 *years*	2-3 *years*	3-4 *years*	4-5 *years*	5-6 *years*	6-7 *years*	7-8 *years*	*Totals*	% *Totals*
Non-paralytic	0	0	4	2	0	0	1	1	0	1	0	9	9·1
Encephalitic	0	0	1	0	1	0	1	0	0	0	0	3	3·0
Bulbar	0	0	3	1	0	1	3	0	0	0	1	9	9·1
Spinal-paralytic	7	2	7	18	12	18	6	5	0	2	1	78	78·8

TABLE V. SPINAL CASES ACCORDING TO AGE AND SEVERITY

	No. of Cases	% *Spinal Cases*	0-5 *months*	6-11 *months*	12-17 *months*	18 *mths*-2 *years*	2-3 *years*	3-4 *years*	4-5 *years*	5-6 *years*	6-7 *years*	7-8 *years*	*Unknown age*
Spinal Cases	78	100·0	2	7	18	12	18	6	5	0	2	1	7
I. Mild	20	25·6	0	1	5	3	6	1	2	0	2	0	0
II. Moderate	4	5·1	1	0	0	1	0	0	0	0	0	1	1
III. Moderately severe	23	29·5	0	4	6	4	5	2	1	0	0	0	1
IV. Severe	18	23·1	0	1	6	3	5	1	1	0	0	0	1
V. Very severe	8	10·3	1	1	1	1	1	2	1	0	0	0	0
Unclassified	5	6·4	0	0	0	0	1	0	0	0	0	0	4

COURSE OF THE DISEASE

Any complete study is notoriously difficult with Bantu patients. Of the 99 patients, 5 died (2 encephalitis, 1 bulbospinal and 2 very severe spinal), 9 were not paralysed nor suffered any disability, and 72 other patients were seen after their discharge from hospital (there was no follow-up at all in 13 cases).

Of 31 patients who were seen 1 month after the onset of their disease, 2 were cured (a mild spinal case and a combined bulbar and mild spinal case), 3 bulbar cases showed slight improvement, and 2 other bulbospinal cases showed improvement of the bulbar palsies only. The remainder were 24 spinal cases who were unchanged.

Of 19 children who were followed up for 2 months, 1 bulbar and 13 spinal cases were unchanged, 2 very severe cases had improved and could now be classified as severe, 2 moderately severe had improved to mild, and 1 initially mild spinal case showed improvement.

Of 22 children followed up for times varying from 3 to 6 months, 18 showed no change, 1 moderately severe spinal case had improved to mild, 2 mild spinal cases showed very considerable improvement though weakness could still be detected, and 1 patient who had been classified as encephalitic and spinal now showed no paralysis but still had very slight nystagmus.

Differential diagnosis illustrated by clinical case notes

As McMath *et al.*[3] point out, poliomyelitis may be difficult or even impossible to diagnose in the *pre-paralytic stage*. Examination at this time may reveal equivocal signs of meningeal irritation with doubtful Kernig and Brudzinski signs and often very slight neck rigidity. They consider the 'tripod' and 'kiss the knees' signs, and the presence of pyrexia accompanied by retention of urine, as valuable diagnostic evidence. However, in children of 3 years and less, as in most of the Baragwanath group, the patient's cooperation is seldom secured sufficiently to elicit these signs. Cases 1-9 were all admitted with diagnoses referable to meningeal irritation, and all gave abnormal cerebrospinal fluids.

Case 1, a male aged 8 months, *case* 2, a male aged 1 year and 3 months, *case* 3, a female aged 1 year and 4 months and *case* 4, a male aged 9 months, all had neck stiffness and high fever and were admitted as cases of *meningitis*. Case 2 became paralysed next day, and later developed respiratory involvement, whilst case 1 was found on admission to have mild spinal paralysis and a bulbar palsy. Cases 3 and 4 did not develop paralysis.

Cases 5 *and* 6 were less acutely ill and were admitted as probable cases of *tuberculous meningitis*. Case 5, a male aged 2 years, presented with a 'sore neck' and had neck stiffness for 2 days in hospital before developing a weakness of the left leg, when it was learnt that a sibling had been admitted to another hospital with poliomyelitis. Case 6, a male aged 4 years, was so uncooperative and apparently hysterical for some days in the ward that he could not be properly examined, and his mild paralysis was not immediately discovered.

Case 7, a male aged 1 year and 4 months, with high fever, was admitted as a possible *encephalitis or tuberculous meningitis* because he was thought in the out-patients department to have an upper-motor-neurone weakness of the facial nerve. After observation in the ward it became clearly lower-motor-neurone in type, and the cerebrospinal fluid showed 119 polymorphonuclear leucocytes and 47 lymphocytes per c.mm., with normal chemical findings.

Case 8, a male aged 1 year and 3 months, was admitted as an *encephalitis*, with neck stiffness and increased cells and protein in the cerebrospinal fluid but without paralysis.

Case 9, a female aged 1 year and 3 months, was admitted as *neck stiffness* for further investigation. On admission she was found to have generalized weakness, and was still very severely paralysed 3 months later

The pre-paralytic stage has been shown to last longer in young infants; the average interval between the date of onset of illness and the first detection of paralysis was 5·5 days among infants under 1 year of age as contrasted with 3·5 days among children and adults.[4] In 5 cases (children) in which the diagnosis is known to have been missed at Baragwanath out-patients, and who later were referred by fever hospitals, the diagnoses on their record cards (just a few days before paralysis set in) varied from pharyngitis to gastro-enteritis. Cases 10 and 11 are examples of pyrexial children in the pre-paralytic stage of poliomyelitis who escaped diagnosis and were admitted.

Case 10, a female aged 3 years and 10 months, had a history of fever and pain in the chest and X-ray evidence of right middle-lobe consolidation. She was admitted with the diagnosis of *broncho-pneumonia*, and next day there was pain in the back with muscle spasm. The cerebrospinal fluid showed 84 polymorphs and 10 lymphocytes per c.mm. and a protein level of 82 mg.% She was referred to the fever hospital, where a mild paralysis ensued. Weakness of the right shoulder-girdle muscles was still present 2 months later.

Case 11, a female aged 4 years, had *abdominal pain for investigation* as her admission diagnosis, and was considered a case of appendicitis by the referring doctor. She had vomited with her severe abdominal pain 3 days before. On admission she was found to have tenderness and pain in limb muscles as well as abdomen and she developed paralysis.

The painful muscles in Case 11 should have indicated the probable diagnosis, for the paralytic phase of poliomyelitis is commonly heralded by tenderness, stretch pain and fibrillary contractions in muscles, with increase of the deep reflexes.[3] The fibrillary contractions in very young patients are usually masked by subcutaneous fat, but the other signs and symptoms sometimes give rise to great difficulty in diagnosis. The increased reflexes caused case 12 to be admitted as an apparent left hemiparesis, and it was only a few days later that poliomyelitis was suspected.

Case 12, a male aged 1 year and 4 months, was admitted with the diagnosis of *hemiparesis with encephalitis*. He had been unable to walk for some days and although he would not use his left arm or stand on his left leg, the jerks were brisker on that side. Two days after admission he developed some doubtful weakness of the right arm, and on the 4th day his left knee and ankle jerks became diminished for the first time. On his 9th hospital day all his reflexes were present and apparently equal, but there was a mild left quadriceps weakness. His cerebrospinal fluid on admission showed 5 polymorphs and 49 lymphocytes per c.mm. with a protein of 77 mg. A week later the cell count was normal, but the protein was still 56 mg. Type-I polio virus was grown from the stool of this patient. He was apyrexial throughout his stay in hospital.

On account of the presence of all reflexes on admission, the following case was not suspected of spinal poliomyelitis.

Case 13, a male aged 1 year and 8 months, with a history of mild diarrhoea and convulsions for 2 days, was admitted in *coma* with marked trismus. Muscle tone was poor and there was no neck rigidity. All reflexes were present and equal. His cerebrospinal fluid contained 130 polymorphs and 95 lymphocytes per c.mm. and was normal biochemically. He died 18 hours after admission, and at post-mortem was found to have changes typical of polio-encephalitis, and poliomyelitis in the anterior horn cells of the cord.

Case 13 is an example of how some cases of polio-encephalitis rapidly become comatose before paralysis can be diagnosed. Such widespread polio-encephalitis is almost invariably fatal.[5] The picture of coma, trismus and convulsions was seen in another in-patient at the time of the poliomyelitis epidemic, and the diagnosis of polio-encephalitis was mistakenly made, for neither laboratory nor post-morten evidence of the disease was found. Both these cases have been fully described elsewhere in a discussion on trismus.[6] This is an interesting and well described sign occurring in poliomyelitis, and attributed to mid-pontine tegmentum involvement at the site of the motor nucleus of the V cranial nerve.

In the paralytic phase of the disease, when *lower motor neurone paresis* develops, diagnosis becomes much easier, but that confusion still arises when the patient is very young is shown by the admission to Baragwanath Hospital of several infants with paralysis. Post-diphtheritic polyneuritis is not uncommon at Baragwanath Hospital and it is easy to understand, therefore, how this diagnosis may be made on an extensively paralysed patient, when seen in the early stages of an epidemic.

Case 14, a female aged 3 years, was admitted as a *polyneuritis* on account of generalized weakness. Next day it was appreciated that in the upper limbs the paralysis was asymmetrical, and the cerebrospinal fluid contained 38 polymorphs and 140 lymphocytes per c.mm. Later Type-I polio virus was grown from her stool.

The amyotonia congenita syndrome due to infantile spinal muscular atrophy may give rise to confusion in the small infant; both conditions result from pathology in the anterior horn cells.

In case 15, a female aged 2 months, 2 convulsions had occurred the night before admission and there was a sudden onset of 'limpness'. The baby was admitted with the diagnosis of possible *amyotonia congenita* or *polyneuritis*. All limbs appeared weak and flaccid, except the right arm, where some muscle tone was present. The right biceps and triceps jerks were the only reflexes that could be elicited. There was some paralysis of intercostal muscles and breathing was chiefly diaphragmatic. The cerebrospinal fluid contained 5 polymorphs and 19 lymphocytes per c.mm. and the protein was raised to 71 mg.%. A month later the patient still had a flaccid paralysis of the left arm, right leg and both glutei.

In the differentiation of poliomyelitis from amyotonia congenita, the history may help, fever and a sudden onset favouring the diagnosis of poliomyelitis. The paralysis is sometimes bulbar and may involve the muscles of respiration in both conditions, but is symmetrical in distribution in amyotonia congenita. Asymmetry in an extensively paralysed small baby may only be demonstrable after careful and patient examination. The cerebrospinal fluid in case 15 was characteristic of poliomyelitis; in amyotonia congenita the cerebrospinal fluid is normal. Final proof is offered by the virus laboratory.

Cases 13 and 15 showed convulsions, but these occur so rarely in poliomyelitis that, in spite of a flaccid left arm in a 5-week-old infant (case 16), the admitting officer who saw him in a convulsion felt that the diagnosis of poliomyelitis was so unlikely that he admitted the patient to the general wards.

Case 16, a male aged 5 weeks, was admitted with the diagnosis *monoparesis for investigation*. His left arm had been noticed to be paralysed for 1 day, and he had been feverish for 2 days. A generalized convulsion occurred just before admission. There was a flaccid paresis of the left arm. Subdural taps were negative and the cerebrospinal fluid contained 29 polymorphs and 48 lymphocytes per c.mm. and 71 mg. of protein per 100 ml. Twelve hours later the right leg was thought to be moving less briskly than the left and the patient was transferred. When seen a month later the arm was still paralysed and some weakness of both legs was present.

In New York in 1949 and 1950 convulsions occurred in only one infant among 92 under 1 year with poliomyelitis.[4] In the two very young paralytic Baragwanath cases, cases 15 and 16, the convulsions may be attributed to 'febrile convulsions', but in case 13 the convulsions were almost certainly due to the encephalitis, and it is known that the encephalitic form of the disease sometimes causes convulsions even in adults.[7]

Only about 1/5th of the paralysed patients gave a clear history of a minor illness, and although it has been said that in about 40% of paralytic cases a minor illness can be distinguished,[5] it must be remembered that the patients were for the most part under 3 years of age, and more than half of these below 18 months, and also that history-taking in a busy out-patient department among a multi-language group is often rather brief. There were some extremely observant mothers who noticed a slight weakness some days before the medical officer was sufficiently convinced to subject the child to lumbar puncture.

In each of these several cases the cerebrospinal fluid showed changes typical of poliomyelitis.

In the diagnosis of paralytic poliomyelitis, pseudo-paralysis may give rise to confusion, and the pseudo-paralysis associated with congenital syphilis is seen not uncommonly at Baragwanath Hospital. The pseudo-paralysis of scurvy, however, is not seen, as scurvy is exceedingly rare in Bantu children, although a common disease in adults.[8] The pseudo-paralysis caused by osteomyelitis was mistakenly diagnosed as poliomyelitis at Baragwanath Paediatric Out-Patients during this epidemic. Some of the out-patients were of special diagnostic interest.

The mother of *case* 17, a male aged 9 months, noticed 'funny movements of the eyes' for 1 day. On examination remarkable coarse nystagmoid movements of the eyes were present, and there was a doubtful weakness of the sternomastoids. The child was cheerful and did not look ill. There were 11 polymorphonuclear leucocytes and 105 lumphocytes per c.mm. in the cerebrospinal fluid. The chemistry was not known until the following day, and the baby was nearly admitted to the general wards with a diagnosis of tuberculous meningitis. However, as this disease rarely occurs under 1 year at Baragwanath Hospital without radiological evidence of gross chest tuberculosis, an out-patient chest X-ray was taken. This appeared completely normal and the child was sent to the Fever Hospital with a diagnosis of poliomyelitis, which proved to be correct. He later developed a lower-motor-neurone paralysis of his left leg. This recovered, but some months later very slight eye movements could still be detected.

These coarse movements of the eyeballs on attempted fixation, known as opsoclonia and differing from true nystagmus, which however is also seen, have been described by several observers.[5, 9] The studies of Baker and Cornwall[10] and Matzke and Baker[11] have shown that the cerebellum and midbrain are frequently implicated in the pathology of poliomyelitis, even although clinical manifestations of such involvement are uncommon.

Two children made their first visit to out-patients with *sores in the mouth* and both were diagnosed as herpetic stomatitis. One became mildly paralysed 4 days later and still had weakness a month later, and one became moderately severely paralysed 9 days later, but then developed involve-

ment of the respiratory musculature in the Fever Hospital and died after a week. It is interesting to speculate whether these children did in fact have herpangina due to the Coxsackie-A virus, which virus is often isolated in proven cases of poliomyelitis.[12]

DISCUSSION

It has recently been suggested that acute poliomyelitis is not infectious and patients can safely be admitted to a general ward.[13] Anyone who has personally seen cross-infection occur would disagree. In 1949 several long-term cases developed paralytic poliomyelitis after the admission of 2 cases of poliomyelitis to their cubicled infants' ward. These patients had diseases such as leukaemia, Wilms's tumour, hydrocephalus.[14] Horstmann *et al.* have said that 'on the basis of virus isolations and serological evidence, poliomyelitis infection may be said to be as contagious as measles among susceptible individuals in a family setting'.[15] Despite great care and the use of diagnostic lumbar puncture in the outpatients department, 18 patients were admitted to Baragwanath Hospital. Of these, 16 have been discussed; the remaining 2 patients were known to have poliomyelitis, but one, a fatal case, had to be admitted as he was *in extremis* and too ill to send the long journey (20 miles) to the nearest fever hospital, and the other was admitted owing to incorrect information about the duration of the disease. No case of cross-infection was noted as a result, but the turn-over of patients in Baragwanath Hospital is extremely rapid.

There was a preponderence of very young children amongst the poliomyelitis cases, and this often made diagnosis difficult. Elsewhere it is generally agreed that poliomyelitis is showing an increasing incidence in the older age-groups. Smith *et al.*,[2] who show an increasing incidence of the non-paralytic form of the disease with age, suggested that the more frequent recognition of these forms is the cause of the apparent increasing incidence in older age-groups. However, an increasing incidence with age has also been found among paralytic forms of poliomyelitis.[16]

In New York, among 92 cases in children less than 1 year old, non-paralytic forms were infrequent, totalling only 9·8%, compared with 44·7% in children aged 5-15 years.[4] In London the percentage of paralytic cases under 1 year was higher than that for any other group.[4] At Baragwanath Hospital, on the other hand, of the 17 babies under 1 year with poliomyelitis, 4 (23·52%) showed no paralysis; only 9 out of the 99 children with poliomyelitis were non-paralytic cases, and 6 of these 9 were under 18 months. In view of their recognition at an early age, it seems unlikely that non-paralytic forms in the older age-groups were missed. Presumably when the Bantu also show a trend towards older age-incidence, the percentage and age of the non-paralytic forms will increase.

Medalie[13] has shown that the age incidence in patients in Boksburg-Benoni Hospital in 1956 was lower among the non-Europeans than the Europeans. This may be due to economic and hygienic factors rather than racial factors. In America, Negroes appear to suffer from this disease as frequently as Whites.[17, 2] The shift in incidence of poliomyelitis to the older age-groups appears greater in the families of higher economic status in America. In these it is seldom seen at all under one year.[18]

The percentage of paralytic poliomyelitis was higher in the Baragwanath series than that reported in most recent epidemics elsewhere, and bulbar forms were fewer.[9, 2, 4, 19-21] Encephalitic forms of poliomyelitis varied in New York in incidence from 2% to 8%.[2, 17] At Baragwanath 3% of the cases were encephalitic.

The severity of the cases was striking. Smith *et al.*[2] found only 35% of their spinal cases fell into the moderately severe, severe and very severe groups, whereas 63% of the Baragwanath cases were in these categories.

The cerebrospinal fluids of the Baragwanath cases did not show any unusual features and the findings, including the occurrence of normal fluids, are similar to those described in most epidemics.[2, 22]

The follow-up of these patients is quite inadequate because, although most authorities agree that the maximum degree of recovery in muscle function in poliomyelitis occurs in the first 6 months, recovery often continues for a couple of years. The most severely afflicted can expect the greatest degree of permanent weakness, and on this basis the young Bantu cases described can be expected to give rise to a considerable number of cripples. The need for early diagnosis, difficult though it may be in this age-group, and for immediate isolation of patients, is stressed.

SUMMARY

In the largest epidemic of poliomyelitis yet seen in the Johannesburg Bantu, which occurred in 1956, 169 cases of acute anterior poliomyelitis were seen in the Paediatric Unit, Baragwanath Hospital, Johannesburg, in the first half of the year.

The clinical presentation, sex, age, form of disease and severity of 99 cases are described and discussed.

The preponderance of very young cases is discussed. They presented special diagnostic problems and made diagnosis difficult; 16 children who initially escaped diagnosis and were admitted to the general wards are discussed.

Non-paralytic forms of the disease were relatively few, and mostly under 18 months. Bulbar cases were relatively few. The severity of the cases was striking.

Some data on the immediately post-isolation progress of the cases is presented.

I wish to thank the Medical Superintendent of Baragwanath Hospital for permission to publish this paper; Dr. E. Kahn, Senior Paediatrician and Dr. S. Wayburne, Paediatrician, in whose wards these patients were observed; and Dr. R. Cassel and staff of the South African Institute for Medical Research laboratory at Baragwanath Hospital.

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PERFORATING OCULAR INJURY: A LONG-TERM FOLLOW-UP*

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Between the years 1946-1955, 1,303 cases of perforating injury of the globe (excluding those resulting from intra-ocular foreign body) were admitted to Moorfields Eye Hospital, London. Of these cases 511 are analysed in this report. This number includes only those cases which were followed up for at least 3 years; no other form of selection was used. Where the out-patient notes were not sufficiently up-to-date, a letter was written to the patient at his last known address asking him to re-attend for examination. In some cases other hospitals kindly supplied recent information on transfer cases. The average period of follow-up was 5 years, 3 years was the minimum, and 10 the maximum. One was surprised at the large numbers who responded and were eager for a fresh examination and progress report. Approximately 10-15 of these patients were found to be suffering from complaints—related or not to the injury—which required further treatment, apart from those still being treated for their original injury.

Right and left eyes were found to have been injured in an equal proportion of cases.

The cases are grouped in Table I according to the origin of the injury. They included 206 children under the age of 12 (40% of cases), injured and 61 of these (30%) had the eye removed; 92 of these children suffered their injury from a bow-and-arrow, scissors, knife or dart (the children constituted almost 3/5ths of these groups) and 32 of these 92 had the eye removed.

TABLE I. CAUSAL INSTRUMENTS

	Iron Metal	*Tool*	*Knife Scissors*	*Glass*	*Dart Arrow*
Eye retained	88	51	48	43	39
Eye removed	19	24	17	10	23
Total	107	75	65	53	62
IOFB (Levy, 1957) ..	65	91	0	4	0

	Wood	*Stone*	*Explo-sion*	*Miscel-laneous*	*Total*
Eye retained	31	16	10	32	358
Eye removed	26	10	8	16	153
Total	57	26	18	48	511
IOFB (Levy, 1957) ..	4	3	9	96	272

Miscellaneous (96) comprises 76 hammer-chisel injuries. IOFB = intra-ocular foreign body.

Operative Technique. Operation at the earliest possible opportunity was deemed advisable, and under general anaesthesia. Direct suturing of corneal or scleral wounds was a routine procedure, and was very frequently done together with a conjunctival flap covering the wound. Where the iris was incarcerated in the wound but not prolapsed and the anterior chamber was re-formed by the sealed wound, the case was usually treated conservatively. My own opinion, however, is that it is desirable to free the uveal tissue in the recent state where the tissue is still not adherent. Re-formation of the enterior chamber is now attempted as a routine measure (although it was not done in most cases in this series) and is usually achieved by the use of intra-aqueous injection of sterile air or saline. This is particularly useful where uveal or lens damage has occurred. Subconjunctival penicillin and streptomycin and Mydricaine were used at operation, and intensive antibiotics post-operatively. If anterior synechiae develop post-operatively, no attempt should be made to divide them for some while—4-6 weeks. Sutures were left *in situ* for 10-14 days.

Out of the total of 1,303 cases of perforating injury 164 eyes were blind or removed, i.e. 12·6%; 11 of these had no light perception. Since the main deciding factor in prognosis is the state of the lens, the remaining 358 follow-up cases are divided into (1) those with a clear lens (138, 27%), (2) those with a localized opacity of the lens (45, 8·8%), and (3) those with cataract (164, 32·1%) (Table II).

TABLE II. VISUAL RESULTS ACCORDING TO STATE OF LENS

Lens	6/9+ No.	6/9+ %	6/12 No.	6/12 %	6/18-6/24 No.	6/18-6/24 %
Clear	83	60·2	13	9·4	12	8·7
Local Opacity	16	35·6	9	20·0	11	24·4
Cataractous	16	9·1	6	3·5	20	11·4
Total	115	31·6	28	7·8	43	12·0

Lens	6/36-6/60 No.	6/36-6/60 %	−6/60 No.	−6/60 %	*Total*
Clear	13	9·4	17	12·3	138
Local Opacity	5	11·1	4	8·9	45
Cataractous	18	10·3	115	65·7	175
Total	36	10·6	136	38·0	358

Lens and Vision. From Table II one can see that almost 1/3rd of the eyes retained had 6/9 visual acuity or better (40% if one adds the 6/12 cases). Where the lens was unaffected 70% achieved 6/12 or better, and if there was only a localized opacity in the lens more than half (55·6%) fell in this group; whereas if a total cataract resulted the final visual acuity percentages were reversed with only 9·1% reaching 6/9 or better (a potential result, for only 5 cases wore contact lenses). Almost 2/3rds of the cataractous eyes had less than 6/60 vision, and in all the retained eyes nearly 40% had less than 6/60. The percentage of cataractous cases in the higher visual categories would be much improved if all those requiring operation for mature cataract or thickened posterior lens capsule were operated on; there were 113 of such cases, or over 2/3rds of the cataract cases.

Removal of Eye. Of the eye removals, 42 were performed on admission, 2 for gross infection and 40 because of gross injury. As will be seen from Table III, 87% of the grossly injured eyes were removed within 2 weeks, whilst in only

TABLE III. REASONS FOR EYE REMOVAL: TIME INTERVAL

	Total	%	*Immediate Removal*		*Removed in 2 weeks*	
Gross injury	74	48·4%	40	54·1%	64	86·5%
Infection	36	23·5%	2	5·6%	8	22·2%
Dangerous	23	15·0%	0	—	0	—
Phthisical	20	13·1%	0	—	1	5·0%
Total	153	100%	42	—	73	—

	Uveal Prolapse		*Cataract*
Gross injury	74	100%	?
Infection	24	66·6%	22
Dangerous	18	78·3%	17
Phthisical	15	75·0%	10
Total	132	—	?

* A paper presented at the South African Medical Congress, Durban, September 1957.

1/4th of the removals which were carried out for other reasons were performed within 2 weeks. Gross injury was the cause of 74 removals, whilst 36 were taken out for panophthalmitis. Of the latter 24 (2/3rds) had uveal prolapse and 22 cataract. There were 23 irritable and dangerous eyes and 20 phthisical eyes removed; 15 (3/4ths) of the latter had uveal prolapse. Sympathetic ophthalmia was only recorded in one case.

Corneal Scarring. In 41 cases (11·5%) corneal scarring was the cause of visual acuity of less than 6/12. Limbal wounds were found to have no bearing on prognosis, and both corneal scarring and limbal wounds were evenly distributed through all levels of vision.

Uveal prolapse was not so evenly distributed. It occurred in 56% of the 511 cases. Of the 138 cases with a clear lens 65 cases (47%) suffered prolapse; of the 83 cases with a clear lens in the 6/9+ group, 31 (37·3%) had uveal prolapse, compared with the 10 cases (58·8%) out of 17 in the −6/60 group. In the cataractous group, 80 (50·9%) prolapses occurred in 175 cases; of the 16 cataracts in the 6/9+ group, 3 cases (18·7%) had prolapsed uvea, compared with 67 cases (57·8%) out of 116 in the −6/60 group. (See Table IV.)

TABLE IV. CASES WITH UVEAL PROLAPSE ACCORDING TO VISUAL ACUITY AND STATE OF LENS

	6/9+		6/12		6/18-6/24	
Clear	31	37·3%	9	69·2%	5	41·7%
Cataract	3	18·7%	3	50·0	7	36·8%
	6/36-6/60		−6/60		*Total*	
Clear	10	76·9%	10	58·8%	65	47·0%
Cataract	9	50·0%	67	57·8%	89	50·9%

Sympathetic ophthalmia occurred in only 3 cases, one of which lost the offending eye and the others were successfully treated with systemic and topical cortisone.

Anterior synechiae were found in 26 (18·8%) cases of the group with clear lens, 7 (15·6%) cases of the group with localized lens opacity and 81 (46·9%) cases of the cataractous group (Table V). In these 114 cases, 55 attempts were made to divide the synechiae—14 were successful. From Table V it can be seen that the more damage to the eye at the time of injury, as indicated by the presence of cataract, the more likely are synechiae to occur.

TABLE V. ANTERIOR SYNECHIAE ACCORDING TO STATE OF LENS

Clear Lens		*Localized Opacity*		*Cataract*	
26	18·8%	7	15·6%	81	46·9%

Medical and Surgical Treatment. Of the 358 cases (Table II) with perforating injuries, 112 cases were treated medically in the first instance and 35 of them (31·2%) developed synechiae (Table VI). Those primarily treated surgically totalled 246, of which 68 had no iridectomy. Of the latter 68, 39·7% (27 cases) developed anterior synechiae, whilst 32·0% (57 cases) of the 178 iridectomized cases did so. (The 'combined series' referred to in Table VI includes the cases in the present survey and a group of cases from a more recent but similar series.) The high proportion of anterior synechiae in the group in which air instillation was performed reflects the fact that at the time of treatment of these cases the use of air was confined to the severely injured cases, usually those with marked lens damage.

TABLE VI. MEDICAL AND SURGICAL TREATMENT AND ANTERIOR SYNECHIAE

	Present Series			*Combined Series*		
	Ant.	*Synechiae*	*All cases*	*Ant.*	*Synechiae*	*All cases*
Medical	35	31·2%	112	50	29·2%	171
Iridectomy: wound closed	40	30·3%	132	47	27·2%	173
Iridectomy: wound not closed	12	33·3%	36	16	27·6%	58
Iridectomy: wound closed: air	5	50·0%	10	5	41·8%	12
No iridectomy: wound closed	27	39·7%	68	—	—	—
Total surgical with iridectomy	57	31·4%	178	68	28·0%	243

Wound closure refers to that done surgically.

More recently the use of air or saline re-formation of the anterior chamber has become a routine procedure.

Localized Opacity of Lens. The group with localized lens opacity proved the most interesting section. The follow-up period averaged 5½ years in this group and the lens opacity, usually not more than a few millimetres large and on occasions in the form of a track right through the lens, showed no tendency to involve the whole lens. The capsular wounds always sealed off straight away. Over one-half (55·6%) of this group of 45 cases retained visual acuity of 6/12 or better. In 4 cases a vitreous haemorrhage prevented the vision from reaching 6/12, whilst in 6 cases retinal scarring (including the macula in 2 cases) interfered with vision.

TABLE VII. LOCALIZED LENS OPACITY AND VISUAL ACUITY

	Total	6/12+		−6/60	
Performing Injury	45	25	55·6%	4	8·9%
I.O.F.B. (Levy, 1957)	49	36	73·5%	5	10·2%
		Vitreous Haem.		*Retinal Scar*	
Performing Injury		4	8·9%	6	13·3%
I.O.F.B. (Levy, 1957)		4	12·5%	9	18·4%

Cataract. Of the 175 cases in which total cataract occurred, 55 were aphakic and 113 (about 2/3rds) required lens extraction or needling, which would improve their vision. Only 9 aphakics were using contact lenses (none of this series had acrylic implants), and thus the visual results recorded for the cataract group were only potential figures. The vast majority of these patients (uniocular cases functionally) had returned to their previous occupations. A large number were doing work thought to require stereopsis and manual coordination with only one eye functioning. Unfortunately, figures are not available concerning the above-named patients. An enquiry into the binocular vision of injured cases in industry would prove most illuminating. Only 22 cataractous cases (12·6%) achieved 6/12 or better and 115 (almost 2/3rds) resulted in less than 6/60, whilst 16 were phthisical and/or had no perception of light. Of the eyes removed (79), other than those removed for gross injury, 49 (62%) had cataract. In the gross-injury group of removed eyes a cataract was often not recorded because of the severe damage or because evisceration was performed and the pathology not investigated, or because (in most cases) delay before removal was too short to allow a cataract to develop. The poor prognosis of cataractous eyes, related to removal of the eye, is probably to a large extent due to the extent and severity of the injury producing the cataract, rather than to the presence of the cataract itself. However, where the lens capsule is torn and the soft lens matter allowed into the anterior chamber, a picture very different from the sealed-wound cataract is common, showing an irritable and inflamed eye.

Vitreous Haemorrhage and Retinal Detachment. Where the lens diaphragm was not opaque, the vitreous was seen to be clouded with blood in 51 cases (14·2%) and retinal detachment was noted in only 24 cases (6·7%). In the group with clear lens, vitreous haemorrhage and retinal damage accounted for 28 cases (20·3%) with visual acuity less than 6/18, whilst only a few of the aphakic cases where a view of the posterior chamber was possible was the defective vision found to be due to vitreous haemorrhage or retinal defect.

Squint. Of the cataractous cases, 43 (24·6%) had a squint, 12 of which had been cured by operation. Of the cases with a clear lens, 3 had a squint, 2 of which were satisfactorily operated on. A secondary rise in tension was noted in 8 eyes, and 8 eyes suffered from phthisis bulbi.

Causes of Visual Defect. Table VIII shows the cause of loss of visual acuity in the 215 cases with less than 6/12.

TABLE VIII. CAUSE OF VISUAL DEFECT IN CASES WITH V.A. LESS THAN 6/12

Corneal Scar		*Cataract*	
No.	%	No.	%
41	19·1	139	64·7

Vitreous Opacities		*Retinal Damage*		*Total*	
No.	*%*	*No.*	*%*	*No.*	*%*
15	7·0	20	9·3	215	60·6

Often in these cases there was more than one cause for the visual defect, and where this was so the major cause was selected. The figures emphasize the effect of damage to the lens.

DISCUSSION

1. Of the total 1,303 cases admitted to Moorfields, 511 in this period were selected for analysis, solely on the basis that a follow-up of at least 3 years was available. No other form of selection was practised. The number of eyes lost was very low, viz. 153 out of the 1,303 admissions (11·7%); cf. Savory (1953)—39 eyes removed in her series (27·8%).

2. Table I, which shows the source of the injury, emphasizes the large number resulting from accidents with knives or scissors or injury from darts or arrows. Tragically, these occurred to a great extent in children (92 out of 127) and were invariably due to inefficient or negligent overseeing by guardians, who could easily have prevented most of the incidents. Of all the perforating injuries, 40% occurred in children, and in 61 cases the child lost the eye.

3. The deciding factor in the prognosis is overwhelmingly the state of the lens, and the visual results are assessed in this relation. A localized lens opacity brings down the percentage with 6/12 vision or better from 70% in a clear lens to 55·6%, and if a total cataract results the percentage falls to 12·6%, this last percentage refers only to potential visual acuity, for few uniocular aphakic cases—5 in all—wore contact lenses. These potential results could be much improved if all the cataractous cases with persistent opacity (over 2/3rds of the cataractous group) were operated on. The total results are good—40% achieving 6/12 or better (cf. Snell, 1945, 30% achieving 20/40 or better) and 32·1% achieving 6/9 or better (cf. Savory, 1953, 31·9% achieving 6/9 or better).

4. Of the eyes removed, gross injury was the cause of removal in one-half and infection in 1/5th. Almost all the grossly injured eyes were removed within 2 weeks. This, together with the intensive use of antibiotics, is probably the cause for the low incidence of sympathetic ophthalmia—a total of 3 cases. Uveal prolapse had occurred very frequently in the eyes removed (85% of cases—Table III).

5. Corneal scarring and limbal wounds seem to play a very minor role in the prognosis. Both in cases with clear lens or cataract, uveal prolapse was very much more frequent in the groups with lower visual result than in the groups with higher visual result. Understandably, persistent anterior synechiae were commoner in the cataractous group. Although the persistence of synechiae was the same in the cases treated medically in the first instance and in those treated surgically, surgery as a prevention against the development of synechiae is not discredited; the figures show the value of surgery in the more seriously affected cases. No estimation of the value of air or saline re-formation of the anterior chamber in the prevention of synechiae could be made in this series because only recently has this become almost a routine procedure.

6. The persistent stability over a long period of localized opacity in the lens, when only a few millimetres in diameter is well demonstrated again (Levy 1957). Over one-half (55·6%) of this group retained 6/12 or better vision (Table VII). The poor prognosis, both for vision and for retention of the eye, in perforating injury causing severe damage to the lens is well shown; only 13·5% achieved 6/12 or better vision—a potential result—and 62% of the eyes excised (excluding those removed for gross injury, where immediate removal precluded the development or the diagnosis of cataract) were cataractous.

SUMMARY

1. In a decade 1,303 cases of perforating ocular injury, not due to intra-ocular foreign body, were admitted to Moorfields Eye Hospital, and of these 511 cases have been followed for a minimum period of 3 years and an average of 5½ years and are here reported. A low percentage of 11·7% (153 cases) of 1,303 cases lost an eye. 40% achieved 6/12 or better vision, and the prognostic effect of damage to the lens is stressed in relation to visual result and to retention of the eye; the damage causes a drop in the percentage of those with a 6/12+ result from 70% in the group with clear lens to 12·6% in the cataractous group. The stability of localized lens opacity is remarked upon.

2. The tragically high number of children who suffer perforating ocular injury, often with loss of an eye, particularly as a result of accidents with scissors, knives, darts or arrows (almost all preventable) is emphasized.

3. The surgical technique as used in Moorfields is detailed and the use of sterile air or saline for anterior-chamber re-formation is advocated.

This work was made possible by a grant received from the Special Funds of the Medical Staff of Moorfields Eye Hospital, London, through the Institute of Ophthalmology, University of London.

I should like to express my sincere thanks to the surgeons of Moorfields Eye Hospital, London, for their permission to publish these records and for the encouragement I received from them; I would particularly mention Mr. R. C. Davenport and Mr. E. S. Perkins.

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A SURGICAL PROCEDURE FOR THE RELIEF OF VITREOUS OPACITIES ('FLOATERS')*

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This is in the nature of a preliminary report; one cannot judge by only 3 cases. However, as cases suitable for operation are few and far between, and as I feel reasonably sure of the method and its results, I publish this paper in the hope that others will try it and perhaps make known their results.

I must stress that this procedure is only for those very bad cases where the floaters seriously incommode the patient, and should preferably be used where the vitreous is degenerate and semi-fluid. It should not be used for those very common cases where there is a solitary floater or a few small ones.

During the last 18 years, when examining eyes after the coagulation operation for retinal detachment, I have frequently thought that the vitreous appeared to be clearer than before. Allowing for the freer spread of the ophthalmoscopic beam of light in the interior of the eye after replacement of retinal balloons, the impression remained that there were fewer floaters of both the coarse and fine type.

I nurse my cases, where feasible, in the position which puts the coagulation area into the most dependent position, and it struck me that perhaps under conditions of nearly complete absence of body and eye movements ('complete rest' and double bandaging) some at least of the more mobile floaters settling by gravitation to the most dependent part of the eye, and alighting on a recently coagulated patch, might have stuck to this area.

Careful and prolonged ophthalmoscopic or slit-lamp observation of a fine 'dusty' vitreous haze, such as may follow a severe cyclitis, will more often than not fail to show any obvious movement or settling of individual particles, except as part of a tremor of the whole vitreous body; there also appears to be no change in the position of these particles in relation to each other. However, the study of any undisturbed suspension of fine particles in a medium of even very fluid consistency, will fail to show any obvious sedimentation to the observer's eye. Sedimentation might take hours or days, or longer to become apparent.

It seemed to me that by treating the eye as a 'pot of vitreous' and keeping it very still, one might ultimately expect the sedimentation of particles in a gel of fairly solid consistency. In a vitreous gel changed (as one must suppose) by disease or other process, one might expect a softening of the whole, or in 'channels', which might aid the downward fall of particles in suspension.

A speeding-up of this gravitational settling might be expected from the employment of some form of centrifugal force. It would make an interesting study to observe the effects of the Royal Air Force's experimental centrifuge on large and easily identifiable floaters.

The difficulty of assessing partial improvement in a fine 'dusty' vitreous haze needs no stressing. Both subject and observer, even if highly intelligent and perceptive, may be liable to errors of unconscious bias. The test of visual acuity (unless improvement is marked) may not be a reliable guide. The most certain method is to use a similar eye in the same or another patient (examined under identical conditions, e.g. of pupillary dilatation, etc.) as a pre- and post-operative control. This I was able to do in one of the cases described below.

A totally different picture obtains in the eye with coarse, mobile floaters in a degenerate, semi-fluid vitreous. Evidence that such floaters settle after a few hours' rest is borne out by the fairly common history of clear vision on waking, and a rapidly increasing turbidity as the eye and body movements stir up the deposit.

DETAILS OF OPERATIVE TECHNIQUE

The conjunctiva is reflected from the limbus below, and the inferior rectus freely exposed. There is no need to cut the muscle. As there is no inter-retinal separation with fluid or ballooning, as in a detachment, the diathermy reaction comes through easily and only light application of current is needed. (I personally favour the unipolar method.) The insulated diathermy point is applied over an area at 6 o'clock in the form of an ellipse tailing off towards the 5 and 7 o'clock meridia. The main patch of coagulation should stretch backwards from the ora serrata to a distance of about 3 or 4 disc diameters and should lie at the most dependent part of the globe (i.e. with the patient in the Fowler or semi-Fowler position). The coagulation should be moderate, but of sufficient intensity to produce a moderate degree of flocculence in the retina. The small amount of retina thus destroyed gives rise to a small scotoma in the upper field and causes no inconvenience afterwards. The conjunctiva is stroked back to the limbus (stitches are not necessary), atropine ointment is instilled, and both eyes are firmly closed with pads and bandage.

The position of the patient in bed now becomes important and movements of the eyes and head should be restricted to a minimum. The Fowler position is generally a comfortable one, and satisfactory from the nursing point of view. A bolster or 'donkey' under the knees prevents sliding down and helps to restrict movement where a surgical bed is not available. The eyes should not be opened for 10 days, the pads being changed at the dictates of comfort and cleanliness. To avoid stiffness of the neck etc., the position of the head and shoulders should be altered slightly, but all movements (active or passive) should be slow, measured, and deliberate.

Case 1

An African female 28 years old, with a rather vague history of several years during which vision failed to near blindness.

Left eye: Shrunken and disorganized—no perception of light.

Right eye: White and uninflamed. Cornea clear. Old shrunken keratitic precipitates (K.P.). A.C. and iris normal except for a few old synechiae. Pupil half dilated with mydriatic. Lens clear. In the vitreous, large coarse floaters and fine debris in sufficiently dense concentration to obscure all fundus details; hazy red reflex only;

* A paper presented at the South African Medical Congress, Durban, September 1957.

movement of eye produces violent and extensive swirling excursions of floaters in a semi-fluid vitreous. *Vision*, counts fingers.

It was felt that operation should be tried as there was little to lose.

Post-operative Examination. On the 10th day the patient's vision had improved to 6/18 and she was immediately pleased with the result. The fundus could be clearly seen. Large scattered patches of fairly old choroido-retinitis could be seen in the posterior and peripheral fundus. Unfortunately, we were only able to follow this case for a week or two; we lost track of her after her discharge from hospital. However, the last view we had of her vitreous, in spite of the persistence of some floaters, contrasted impressively with the pre-operative appearance, which one of us in the Clinic had likened to a 'dirty tidal pool'.

Case 2

A nun (qualified nursing sister) aged 39. First seen by me on 15 January 1953. The left eye had 'gone blind' some months previously and she had received treatment by two eye specialists at another centre. She was told that she had an embolus from a mitral stenosis. At one time she had an acute left frontal sinusitis, since cleared. To leading questions she remembered that the eye had been inflamed and tender on pressure.

Right eye: Normal. Vision about 2.25 S=6/6.

Left eye: White. No K.P. No synechiae. Fine haxy cloud of particles spread evenly through vitreous. Disc hazy, and suggestive of recent papillitis (? aftermath of retrobulbar neuritis). Vision about 1·0 S=6/24.

X-ray of teeth and sinuses negative.

15 *July* 1954. The patient consulted me again with the history that both eyes appeared to have deteriorated. In view of what I had suspected in regard to her teeth, she had independently consulted a maxillary surgeon who had found a 'diseased chip' of bone which he had removed.

Right eye: White. New floaters and vitreous haze of moderate degree. Haziness and slight swelling of disc suggestive of papillitis of retrobulbar neuritis. Vision about 2·25 S=6/6.

Left eye: White. Great increase of vitreous haze and fine particles since last seen 18 months ago. Vision about 1·25 S=6/18.

17 May 1955. She saw me again and complained that the floaters in the left eye were 'getting on her nerves'. She consented to undergo what I explained to her should be regarded as an experimental operation of reasonable safety. Here seemed to be an ideal case for trial for the following reasons:

1. Her intelligence and willingness to cooperate.
2. The dusty, particulate and apparently immobile nature of the opacities in the vitreous of the left eye.
3. The presence of opacities of a similar appearance in the right eye (but much less so) which would give the ophthalmoscope a picture for comparison. She was not particularly conscious of the floaters in this eye and the subjective haziness of 1954 had disappeared.

I took particular note of a constellation of 3 course moderate-sized floaters in the eye to be operated on. These were the only coarse ones and their position was slightly to the nasal side of the visual axis.

The patient's behaviour after operation was exemplary and her movements in bed must have been minimal.

Post-operative Result. After 10 days the patient was quite emphatic that the vision of the left eye had cleared remarkably. I tried to suggest that possibly she was being deceived partly by wishful thinking and partly by the apparent increased clearness of vision which most patients experience on having their bandages removed. Two days later, however, she was more certain than ever, and now complained for the first time of being conscious of floaters in the right eye, which she now maintained was 'worse' than in the eye operated on. To my surprise and gratification I was able to confirm this ophthalmoscopically. There was still some haze in the left vitreous; but there was no doubt that comparison showed the haze in the right eye to be denser than in the left. There was no reason to believe that since operation on the left eye the haze in the right eye had increased. The appearance was either that my 3 'marker' floaters in the left eye had fused into one, or that two of them had disappeared. The shape of the solitary one appeared different and larger, but its position was unaltered. The visual acuity of the left eye (still partly atropinized) on the 14th day was unchanged (vision about 2·5 S=6/18) but the eye appeared to have become a little more myopic.

The following is an extract from a letter from the patient 3 weeks after operation. 'My own observation of the left eye after operation: Eye feels still a little heavy. Central vision clear. On looking downwards I can recognize the floaters partly, which disappear when I raise the eyeball and look straight ahead. I can only notice the floaters very slightly when turning the eye to the left. I only realize now how much the right eye is affected, as the picture before the right eye is quite different from the figures that floated in the left eye, and have now disappeared. Sister typed the report for me because the eye still becomes tired.'

Case 3

A few months ago this elderly Coloured man returned to me after an absence of 3 years. He had lost the one eye previously, and first came to see me with the request that I should remove his remaining eye, because he was much disturbed by the movements of large dark 'shadows' in the eye. Mentally he did not appear to be normal, and he seemed to be obsessed by his eye. The eye was quiet. The vitreous was a mass of floaters and the fundus details were invisible. Vision, hand movements.

I refused to excise the eye, but offered to perform an operation which might help. It was very difficult to determine how much coagulation one was producing below, owing to the turbidity of the vitreous.

On about the 8th day, the patient had a mental storm and discharged himself from hospital before I could examine the eye.

On seeing him again recently I was astonished to find a relatively clear vitreous and a vision of 6/24 in a relatively contrite and not ungrateful patient.

DISCUSSION

The success of operation where the vitreous is degenerate and semi-fluid and the floaters freely mobile is understandable from an appreciation of simple mechanics. On the other hand, in the presence of what *appear* to be fixed floaters, any clearing of the vitreous might, I submit, be due to the following possibilities:

1. That the structure of the vitreous gel is not stationary. Little is known of its structure in life, or of any flow, circulation or movement that might take place, but it seems possible that some of its particles might *eventually* settle, however slow the rate of sedimentation, if encouraged to do so. Using the analogy of the toy 'snowstorm' in the glass sphere, one can visualize the experiment of gradually increasing the density of the medium in which the 'snow' lies suspended, and eventually producing a state of semi-solidity. Under these conditions one might reasonably expect the 'snow' to take months or even years to settle.

2. The heating effect of the diathermy reaction might have some chemical or physical effect (on its specific gravity?) on the vitreous in the neighbourhood of the diathermy reaction, or on the vitreous as a whole. Anyone familiar with retinal detachment operations will bear witness to the considerable rise in the pressure in the eye after several applications of diathermy. I have always considered this to be due to a swelling of the vitreous gel, i.e. an increase in volume which might be expected to decrease its specific gravity. Here the views of a physicist would be of value.

3. The possible speeding up of a possible vitreous 'circulation', on the analogy of the increase of molecular activity when water or any fluid is heated or put under pressure.

4. The vitreous in the vicinity of the diathermized retina might be made more 'sticky' or adhesive to any falling opacities.

5. The processes of inflammation and repair initiated by the electrical burn might multiply and speed up the rate or

volume of cellular and leucocytic scavenging in the eye. The nearer the debris is brought to the inflamed area, therefore, the better.

My thanks are due to Dr. B. Bromilow-Downing, Superintendent of the Frere Hospital, East London, for permission to publish details of case 1, and to Sister Corona of the Peddie Hospital for her cooperation.

TRAUMATIC HAEMOMEDIASTINUM: A CASE REPORT

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Traumatic mediastinal haematoma is a rare condition. Endress[1] apparently came to that conclusion, for he wrote: 'Search of the literature proved almost fruitless. One brief extract of such a case was found in the October 1916 issue of this journal (American Journal of Roentgenology—Korner[2]). Two other isolated case reports were by Duyster[3] and Schmitt[4] respectively. Horn[5] described a further case.' Endress[1] continues: 'Personal enquiry was made of several radiologists known to be particularly interested in chest lesions, but none could recall seeing a haematoma of the mediastinum from trauma.'

Other cases described in the literature and not mentioned by Endress[1] are those of Eiselsberg and Gold (1931),[6] Zimmermann (1936),[7] Vincent *et al.* (1953),[8] and Laforet (1955).[9]

The aetiological factors have varied considerably. Horn's case[5] was caused by a stab wound penetrating into the chest. The cases of Eiselsberg and Gold[6] were due to fractures of the thoracic vertebrae. Both Schmitt's case[4] and Duyster's case[3] were due to multiple chest injuries, whilst Endress's cases[1] were caused by 'steering-wheel' automobile accidents. Zimmermann's case[7] was due to lateral compression of the thoracic cage. Kerley (1957)[10] has stated that he has seen the condition as a result of operative trauma to the neck in cervical sympathectomies, whilst Kessler (1957)[11] mentions a mediastinal haematoma found post mortem after a carotid angiogram.

CASE REPORT

A young adult Bantu male from whom an adequate history could not be obtained because of language difficulties, was thought to be suffering from injuries to his thoracic cage. He was not dyspnoeic, but appeared to be very shocked. He was not in great pain.

X-ray Examination

The first X-ray of the chest was taken on 8 January 1957 (Fig. 1). Radiographs of ideal diagnostic quality were difficult to obtain because the patient could not be made to understand that he should stop breathing; nor was he able to stand erect with any ease. However, on the available postero-anterior radiographs there was obviously gross widening of the mediastinal shadow. There was, in addition, an opacity occupying part of the middle third of the left hemithorax, which on the lateral view was seen to be situated in the anterior basal segment of the left lower lobe and in the left lingula.

On the basis of these findings a diagnosis was made of mediastinal haemorrhage, together with pneumonic consolidation of the affected lingula and lower lobe segments—the latter it was thought, being in the nature of a 'contusion' pneumonia. As a result of this diagnosis the patient was examined in minute detail and a tiny (2 mm.) puncture wound was found on the left side of the neck over the sternomastoid muscle about 1 inch above its insertion. Subsequently an interpreter was found who spoke the patient's dialect and from whom we learned that this wound was caused by a 'bicycle-spoke' with pointed tip—a not unusual weapon in these parts!

X-rays (Figs. 2-5) taken during the next 10 days showed rapid and progressive diminution of the width of the mediastinal shadow and almost no change in the region of consolidation in the middle third of the left hemithorax.

The opaque left lingula cleared rapidly thereafter, and the lower lobe somewhat less rapidly but just as definitely.

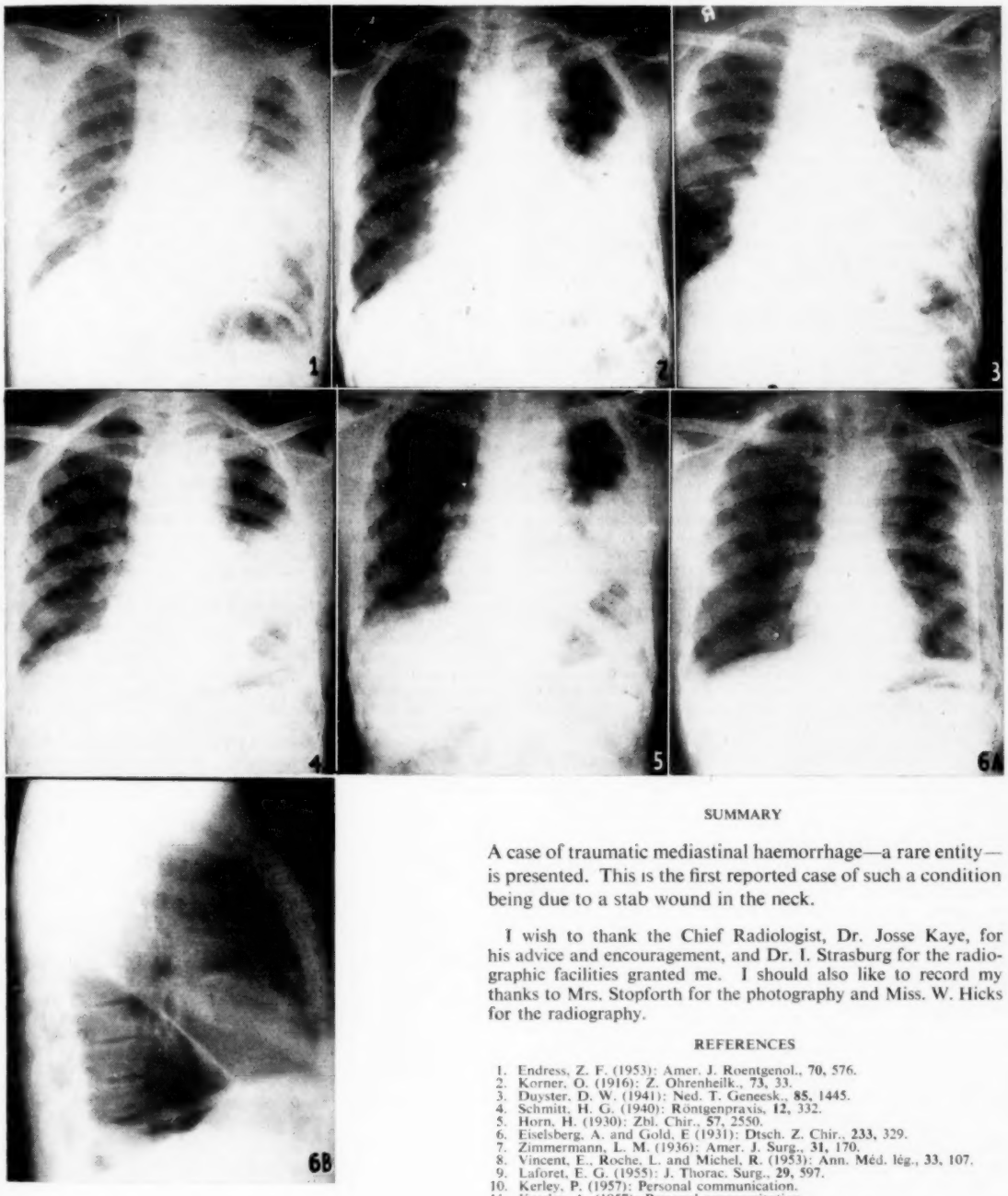
When the patient was discharged on 4 February 1957 (Figs. 6a and 6b), except for some slight widening in the region of the aortic knuckle and in the extreme left apex, the condition had returned almost to normal.

DISCUSSION

These radiological findings are not dissimilar to those of Zimmermann's case[7] in which at first 'the mediastinal shadow was markedly widened' and later 'there was gradual absorption of the mediastinal haemorrhage with residual increase in breadth and density of the right hilus shadow from organization of the haematoma'. Endress's case 1 showed lateral bulging of the superior mediastinum to the right, with a sharp border; 18 days later the radiograph appeared normal.

Duyster's case[3] showed lateral bulging on the left side. Golden (1950)[12] described mediastinal haematoma as a condition in which there is a 'straight and sharp border to the widened mediastinum and if much blood accumulates the pleura tears and blood is then in the pleural space'.

It is felt by both Endress[1] and Laforet[9]—and in this I agree—that many more cases of mediastinal haematoma have occurred than have been reported in the literature. One of the reasons for this suspected frequency is the number of 'steering-wheel' injuries. I agree with Endress,[1] who felt that 'one reason for lack of reports is that films of injuries to the chest are usually done with Bucky technique at short distances and the mediastinum is ignored because of the expected distortion'. In addition, I feel that in all suspected injuries to the thoracic cage or neck a postero-anterior radiograph of the chest should be done as a routine measure.



Figs. 1 to 6

SUMMARY

A case of traumatic mediastinal haemorrhage—a rare entity—is presented. This is the first reported case of such a condition being due to a stab wound in the neck.

I wish to thank the Chief Radiologist, Dr. Josse Kaye, for his advice and encouragement, and Dr. I. Strasburg for the radiographic facilities granted me. I should also like to record my thanks to Mrs. Stopforth for the photography and Miss. W. Hicks for the radiography.

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AS OTHERS SEE US*

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In our leisure hours most of us read novels and some of us read plays; and it may be entertaining for a while to see ourselves as others see us, and recall some of the things said about doctors by novelists and playwrights.

Doctors, like priests and naval officers, belong to a specialized and dedicated profession. Willy-nilly, we are cast in a mould, and inevitably view the layman's opinions with a critical and sometimes prejudiced eye. The sailor, seeing a sea-picture, may have his seamanly sense offended by some technical error. We, too, tend to demand a high standard of medical knowledge from those who write about doctors. In this we sometimes err, for the artist presents a picture of the profession which most patients would readily recognize.

Laymen are frequently infuriated by what they call the 'orthodoxy' of the profession. It is possible that many of us lack self-criticism and are too prone to accept current teaching as permanent truth. Some of our critics seize on this conservatism and exaggerate it.

I suppose that most of us have seen the film of the 'The Robe'. When I was at No. 5 General Hospital in Cairo, all the medical officers read the book. Its message is religious; the narrative is fluent and compelling; and it seems to be the fruit of a good deal of historical research. I should like to quote one incident, towards the end of the book, when the Christian slave Demetrius is brought desperately wounded to the house of his master. His life is despaired of. A physician, Sarpedon, is called in, applies hot fomentations and gives a bad prognosis. Finally, St. Peter, who is in Rome at the time, sees the patient, is closeted with him in prayer, and leaves him on the road to recovery. Sarpedon's fury knows no bounds, and he threatens to expose to the authorities this trifling with Christian seditionists. His real fury is due to his replacement and to the irregular nature of the cure. He is sharply rebuked by Tribune Marcellus. 'You and your Hippocratic oath! You are supposed to be interested in healing! Has it come to pass that your profession is so jealous and wretched of heart that it is enraged when a man's life is restored by some other means than your futile remedies?' Is this really a Roman tribune speaking, or is it the twentieth-century author? I suspect that it is both.

If we now take a forward leap of several centuries and visit the sick-bed of Richard Coeur-de-Lion, dangerously ill, we find a Crusaders' camp whose regular doctors are Jews practising the medicine of the time. The king's faithful right-hand man decides to call in a Moorish physician, el Hakim Adonbec. Misgivings are allayed by trying the new treatment on the dog, in the person of a humble squire, who justifies the experiment by his recovery. The Moor feels the king's pulse. The king, who knows a thing or two, feels the Moor's and announces his satisfaction with the words, 'His blood beats calm as an infant's. So throbs not theirs who poison princes'. El Hakim gives the king an elixir made by dipping a silken bag into a silver goblet containing fluid. This pharmacological tea-drinking throws the patient into a sleep of several hours, from which he is awakened cured, by having an aromatic sponge thrust under his nose.

In this story, which is from 'The Talisman', by Sir Walter Scott, we are shown a healer whose ethical standards are austerely rigid. He will not discuss the case in the sick-chamber. He refuses to protect himself by consultation with Christian bishop or Jewish doctor, although the penalty of failure is a violent death. And finally he disdains the king's offer to empty his coffers with the words, 'The medicine would lose its effect, did I exchange it for gold or diamonds'—a reason for *pro deo* treatment not commonly accepted today.

William Shakespeare took his plots from many sources, and I cannot recall any specific preoccupation with doctors, but he has given us some wonderful clinical descriptions. His lines about Juliet's trance are irresistible, although I must confess that it was Friar Laurence and not a doctor who administered the drug.

'Take thou this vial, being then in bed
And this distilléd liquor drink thou off;
When presently through all thy veins shall run
A cold and drowsy humour, for no pulse
Shall keep his native progress but surcease;
No warmth, no breath, shall testify thou liv'st,
The roses in thy lips and cheeks shall fade
To paly ashes; thy eyes' windows fall
Like death when he shuts up the day of life;
Each part deprived of supple government
Shall, stiff and stark and cold, appear like death;
And in this borrowed likeness of shrunk death
Thou shalt continue two-and-forty hours
And then awake as from a pleasant sleep.'

—A horrifying picture of death on the table narrowly averted. But what would an anaesthetist not give to be able to master so accurate a dosage?

We do, in the end, get our doctor. Romeo, believing Juliet to be dead, persuades a miserably poor apothecary to flout the Dangerous Drugs Act of that age and sell him a scheduled poison:

'Noting this penury, to myself I said
An if a man did need a poison now,
Whose sale is present death in Mantua
Here is a caitiff wretch would sell it him.'

'My poverty, but not my will consents' says the Apothecary, and promptly sells him the poison. Romeo pays him with the half-contemptuous phrase 'I pay thy poverty and not thy will'—but here, I fear we are approaching perilously near to the subject of Medical Economics.

Shakespeare conducted no vendetta against doctors, but Molière did. He did not like them at all; and, if the samples of our profession found in his plays truly reflected the state of affairs at the time, I do not blame him. Grave portentousness, fake latinity, and a doctor's hat and gown, ensured the acceptance of a most undistinguished imposter in 'Médecin Malgré Lui', and, indeed it looks as if the author saw little difference between the real doctors and this doctor-for-a-day. The fun in 'Le Malade Imaginaire' is fast and furious, with a posse of doctors battening upon a hypochondriac so devoted to pills, potions, colonic irrigations, general cossettings and useless multiple treatments that he actually plans to marry his daughter to the newly-qualified son of his physician, so as to have a doctor in the family. The refreshing good sense of the brother, Béralde, probably reflects the views of Molière himself. When ill, he says, do nothing. Nature will probably cure you. Doctors can discuss the disease in Latin, name it in Greek, and debate its site and nature for hours, but they cannot cure it. Most patients, in fact, die of their remedies and not of their disease.

This revolt against a system has probably its parallel today. A correspondent in the B.M.J., not long ago, described a new but prevalent disease—'pseudo-scientific meticulosis'. He particularly objected to a certain type of graph, to which he referred as 'those damned dots'. He overstated his case but it must be admitted that we are sometimes so overwhelmed with laboratory data that only the most perceptive can see the wood for the trees.

I once contributed to the *Waste Paper Basket* of the Owl Club, and I called my paper 'Quack! Quack! or one Quack to another'. I made merry for my allotted time with all the queer pseudo-medical cults I could remember and rather rashly tried to draw conclusions. The successful charlatan, according to these conclusions, owed his success to four things; unbounded confidence, ignorance of medical truths, unfamiliarity with statistical methods, and the natural tendency to spontaneous recovery of almost all ordinary diseases. I think I was rash, for the first and last of these help the doctors as much as they help the quacks, and the statistical method has so many pitfalls that only the experts can really use it well.

Bernard Shaw seized on this fact as one of his arguments in that diatribe against doctors, the preface to 'The Doctor's Dilemma'. I like the play well enough. It has among the cast an expert on tuberculin, an old-fashioned physician, a surgeon who cuts out

* Valedictory Presidential Address delivered at a meeting of the Cape Western Branch, 30 January 1958.

nuciform sacs, a fashionable humbug, and a G.P. turned specialist who despises G.Ps. These pieces are moved on the board with unerring Shavian stagecraft. But the Preface is the thing. In it we are told that doctors have no honour and no conscience, other than that possessed by the average Englishman—sentimentality and an intense dread of doing anything that everybody else does not do. We are told that doctors perform unnecessary operations and manufacture and prolong lucrative illnesses; that doctors are liars, are unscientific, and are cruel voluptuaries who torture animals for sheer curiosity. We are told that the surgeon keeps his self-respect only because he does a very bad thing supremely well. All this is written with a passion, a conviction and a casuistry that command respect because they are so difficult to answer effectively.

Shaw liked doctors. He felt proud to call them his friends. He appreciated their hardships and admired their generous qualities. He realized what a struggle most of them had to face. He knew they had to keep up appearances on incomes which were not only inadequate, but capriciously paid. In fact, he went so far as to call them 'hideously poor', which is certainly an exaggeration. This brilliant, malicious, kindly and inexplicable genius has taken us to task. For our own edification—but certainly not for public use—we should formulate a reply. It would be amusing to see the embryo doctor forced to write, as part of his training, a critical analysis of the Preface. What a mess he'd get into!

Shaw felt that doctors were kindly men turned callous by professional custom. Kindness, gentleness, consideration and humanity have long been the traditional attributes of the physician, and lapses in respect of humanity are not readily forgiven by the public. Readers of the B.M.J. will remember Lettsom's bitter accusations against Mark Akenside, the physician-poet. A certain hardness must, of necessity, have been part of the surgeon's equipment in pre-anaesthetic days, but many genuine anecdotes of great surgeons record not only their skill and dexterity, but their humanity.

It is not so with Pierre La Mure's new description of a cataract operation performed on Johann Sebastian Bach. Poor old Bach, near the end of his life, is subjected to the most excruciating tortures. The whole horrid set-up of pre-anaesthetic surgery is seen at its worst. The four brutal half-drunken assistants who strap Bach to a table have already partaken of the potent liquor designed to diminish the patient's pai.. The mincing entry of the pomaded doctor, his incessant chatter about his successes, and the operation itself, with its attendant muffled shrieks, groans and gurgles, seem a nightmare. The reluctance of the patients of those days to undergo operations is easily understood. Perhaps the author of 'Beyond Desire' is unfair to Doctor Taylor, who successfully removed Handel's cataract. In Bach's case the doctor did not merely fail. He ruined his patient by taking his life savings, and Bach's widow died in great poverty.

I see that I have climbed into the twentieth century and then gone back to Bach, and I feel that I now owe it to you to return with all speed to modern times and finish my address. But I cannot resist a passing glance at three novelists, Jane Austen, Charlotte Brontë, and Charles Dickens. The nineteenth century saw great social and industrial changes and the awakening of medicine and surgery. It saw the birth of anaesthesia, the discovery of bacteria, and the beginnings of asepsis.

Jane Austen's doctors had none of these advantages. Her humble apothecaries, of little social consequence to the country gentry whom she described with her quiet satire, were, nevertheless, consulted and respected. They did their work according to their lights and their patients hung upon their rather ambiguous pronouncements. Charlotte Brontë is a little more critical, with a good eye for a humbug, but I remember that Jane Eyre, a neglected and illtreated child, heard almost her first kind words from Mr. Lloyd, the apothecary. Besides being cruel and unsympathetic, Jane's aunt was a snob. The apothecary was good enough for Jane and the servants. When she or her children were ill, she sent for a physician.

Charles Dickens and his doctors could monopolize a whole presidential address. In his times conventional vice opposed conventional virtue, and the doctors were on the side of the angels. Even the workhouse doctors had more humanity than one would expect. I have a few favourites, notably Mr. Losborne, who risks his professional reputation to save Oliver Twist from the Bow Street runners, little Mr. Chillip whom Betsy Trotwood beats with her bonnet because David Copperfield is not born a girl, and the lively drunken Guy's student, Bob Sawyer. It seems that Dickens could not bear an incurably bad doctor, for Bob Sawyer ultimately gave up drink, and led a useful life. The acme of professional virtue is reached in Allan Woodcourt of 'Bleak House', who marries Esther Summerson, the heroine, and spends his life, beloved by all, in that state of benevolent activity and modest prosperity—not wealth— which Dickens felt to be the true status of the profession.

Medical authors provide my last two selections, both dealing with fairly modern industrial practice in Britain. 'The Citadel' we all know. Cronin is a great story-teller and I have sometimes felt that he sacrifices truth to art. I found that I liked the book so much better on a second reading that I had to revise most of my earlier impressions, feeling that the story was on the whole a moving defence of integrity. 'My Brother Jonathan', by Francis Brett Young, seems to me to capture the medical atmosphere much better. A contrast between Jonathan, a doctor by vocation, and the brilliant Harold, who chooses medicine for want of anything better to do, brings me to my final point.

Who should become doctors, and who make the happiest doctors? The novelists and the public plump every time for the man with a vocation. They are right. The devotees, who have never wanted to be anything but doctors, are obvious round pegs in round holes, but there is a second group, whom the discipline of our professional life has gradually moulded. These two groups, and not the Wealth-seekers, the status-seekers, or the indifferent, will enjoy the practice of the most fascinating profession in the world.

FERDINAND VON ARLT AND ERNST FUCHS*

TWO REPRESENTATIVES OF THE VIENNA SCHOOL OF OPHTHALMOLOGY

ALEXANDER JOKL

Johannesburg

The second half of the 19th century and the beginning of the 20th were times of splendour and fame for the medical faculty of the University of Vienna. Many of its professors (Billroth, Brücke, Freud, Hebra, Hyrtl, Lorentz, Nothnagel, Politzer, Rokitansky, Schauta, Skoda, Wertheim and others) became world famous. This was no accident but was the result of the way in which the members of the medical faculty were chosen and the high standard that was deliberately kept up. The most important quality of a professor in Vienna was always considered to be his ability to do original research work. He had to be a pioneer in his branch of medical science, besides fulfilling the requirements necessary for a good teacher. He had to master his speciality in all its branches and ramifications, including laboratory techniques and accessory sciences. He had to be *the* expert, and it was expected of him to have absorbed all that was said and written in the past, and to keep himself informed about current research work done in the laboratories, clinics and hospitals throughout the world. He had to be a good speaker and to be able to lecture freely, without the help of a manuscript. Finally, only men of outstanding character were chosen, teachers who inspired the students not only through their teaching, but by their devotion to duty and their exemplary way of living. Nothnagel, in his inaugural lecture as professor of medicine in 1882 said these words: 'Knowledge gets its ethical value and its true significance only through the spirit in which it is used. Only a good man can be a good doctor.' In the choice

* A paper presented at the South African Medical Congress, Durban, September 1957.

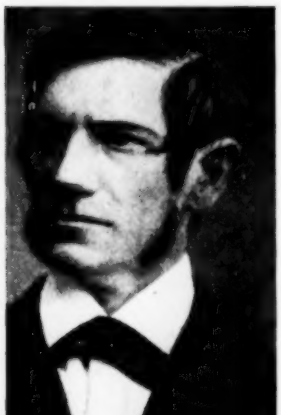
of professors, national considerations were regarded as of secondary importance. If no suitable candidate for a vacant chair could be found in Vienna, a man from another Austrian province or another country was chosen. Professors in Vienna came from Germany, Switzerland, Holland, Italy. It hardly ever happened that a call from the Vienna University was refused. To become a member of this illustrious *collegium academicum* in Vienna was regarded as such an honour that anyone was proud to belong to it. Arlt and Fuchs might be regarded as typical representatives of the Vienna medical school of this period.

FERDINAND VON ARLT

Arlt, born in 1812, son of a poor blacksmith in a small village in northern Bohemia, suffered many hardships during his youth. In his autobiography he tells of the hard work he was put to as a child, of the poor food and the severe cold he suffered during the winter months. During his grammar-school days he lived in the house of his father's half-brothers, whose wife ill-treated him like the proverbial stepmother. His high-school days, in the small town of Leitmeritz, were not much better. He froze and he starved, and he succeeded in keeping body and soul together only with the help of meals to which he was invited by charitable people, and by helping younger pupils in their studies for a small fee.

His family wanted him to become a clergyman, but he decided in favour of medicine and studied at Prague, still not without hardship. After he qualified he became assistant (1840) and later successor (1847) to Johann Nepomuk Fischer, the professor of ophthalmology there. Ten years later (1856) he was called to Vienna, where for 27 years he occupied the chair of ophthalmology treating thousands of patients, operating, teaching and writing. In 1883 he retired from the clinic and in 1887 he died.

Arlt's fame as a scientist rested originally on his text-book, which appeared in 3 volumes between 1851 and 1856. Though more than a hundred years have passed since its first appearance and many changes have occurred in the conception of eye diseases and their treatment, it can be read with interest even today. For the first time clinical facts were strictly correlated with anatomical and physiological principles. What impresses one most are the numerous case histories which illustrate nearly all the diseases described. They bear witness to Arlt's excellent power of observation and his ability to describe what he had observed in clear, simple language. The main weight of the book rests on the presentation of the external eye diseases. The ophthalmoscope had just be invented (Helmholtz, 1851) and many findings made with its help could not then be properly interpreted.



Ferdinand von Arlt

Of Arlt's discoveries only two will be mentioned here: He was the first to show that in higher degrees of myopia the posterior segment of the eyeball was dilated and extended, while the front part was not altered in shape; and he explained correctly the nature of staphyloma as scar tissue formed from the prolapsed iris. He was the first to state in clear words that sight-testing and the determination of the refraction of the eye belongs to the ophthalmologist.

When O. Becker wrote in Arlt's obituary that, in a sense, all the ophthalmologists then living were his pupils, he referred to his *Ophthalmic Surgery*, which appeared in 1874, as a chapter in Graefe-Saemisch's *Handbook of Ophthalmology*. This made him famous throughout the world. For over 20 years most ophthalmologists, wherever they lived, studied it. The historian Hirschberg writes: 'Every one of us, whose duty it was to perform operations on the eye, studied this work with great diligence and consulted it again in every difficult case.' Every line in it is based on the personal experience of the author, collected during 25 years of intensive work. At that time it was the best book on ophthalmology and it remained so for many years.

Fuchs gave a graphic description of Arlt as an operator. Cocaine was then unknown and general anaesthesia was not used in Vienna for eye operations. In cataract operations the incision caused little pain but the iris, being very sensitive, could usually not be brought back to the right position and results without adhesions or prolapse were a rarity. At that time nothing was known about asepsis. Arlt advised putting Daviel's spoon into the mouth to moisten it with saliva before introducing it into the eye. The instruments were washed after each operation, not before. The post-operative treatment was cruel; for 6 days the patient had to lie motionless on his back. Men had often to be catheterized, and the pain and the pressing when trying to pass water often led to bursting of the wound, which of course was not stitched in those days. Hypostatic pneumonia was common, and so was sudden death from pulmonary embolism due to thromboses in the veins of the legs.

Arlt looked like a schoolmaster, and he was indeed an excellent teacher. He sat on a little three-legged stool, surrounded by his pupils. Every case was examined by one of them and was afterwards discussed. Arlt himself writes that he never saw the cases beforehand, and that his diagnosis was based on the observations he made while the student was examining. When teaching young doctors to operate, he assisted them himself; he did not criticize or interrupt them during the operation but after its conclusion discussed their mistakes and the way to avoid them. The teaching of certain branches of ophthalmology, such as ophthalmoscopy, perimetry and refraction was delegated to certain assistants who gave special courses on these subjects—a practice continued in Vienna up to the present day.

Arlt's private practice in Vienna was enormous and we have a good description of it from the pen of Dr. Hans Adler, who for some time was his private assistant. No fee was asked for; everybody gave what he wanted, many nothing at all, even for treatments lasting for weeks and months. His guiding principle was: '*Primum humanitas, alterum scientia*'. In his autobiography he enlarges upon this: 'The real disciples of our art', he writes, 'should not bother about income; this arrives in the course of their activities by itself. Their aim should be to help through science and ability and where these prove insufficient, through compassion and pity for the lot of the sufferer'.

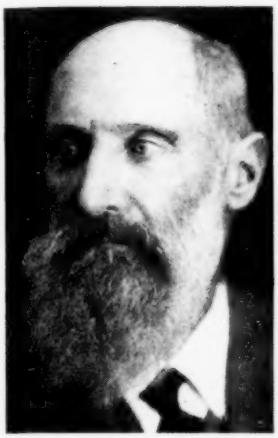
This life, entirely devoted to healing, learning, teaching and research, was not to end without great suffering. Though he was well off, he lived parsimoniously; he did not possess a carriage of his own, but used the horse-drawn trams which were the main means of communication in Vienna at that time. Jumping off from the running car, he one day fell and broke his left arm close to the shoulder. Thrombosis in his left foot supervened, causing severe pain, which did not leave him until his death nearly 9 months later. He suffered amputation of his foot, exarticulation of the fibula, amputation of the thigh, and finally resection of the sciatic nerve. Sedatives became useless to ease the pain and often he implored his doctors to let him die. On 9 March 1887 hypostatic pneumonia ended his sufferings.

ERNST FUCHS

The Fuchs family also came from Bohemia. They had been peasants in the Bohemian Forest, but Ernst Fuchs' grandfather migrated to Vienna and settled there. Ernst's father, a professor in a high-school in Vienna, though not well off, was able to give him an excellent education. As a result of this difference in early education, while Arlt, in spite of his achievements, remained in certain respects restricted in his general outlook, Fuchs had many varied interests and hobbies. He was typically Viennese. He spoke German, French, English and Italian equally well. He was a great reader, and fond of classical art; he loved walking, cycling and mountaineering, and became one of the greatest travellers of his time. Extracts from his travel diaries have been published by his son. He was asked to lecture in far-off countries, and his pupils all over the world often called him in for consultations and operations. He visited nearly all the European countries, Asia Minor, Ceylon, Thailand, Java, China and Japan. With Robert Koch he travelled in Central Africa, and on another occasion he

was invited to organize an eye clinic in Addis Ababa. Lecture tours took him through a great part of the American continent, both north and south.

Fuchs' parents lived in a four-roomed flat in one of those gloomy middle-class apartment houses of Vienna. He went to a gymnasium which had been founded by clergymen from Scotland, one of the best in Vienna. The main subjects of instruction were Latin and Greek. Pupils were expected to work hard, and Fuchs recalls how for many months he started at 4 a.m. In 1868 he entered the medical faculty of the Vienna University. His teachers included Hyrtl, Brücke, Rokitansky, Hebra, Billroth and Arlt. While still a student he showed his ability for research in the physiological institutes both in Vienna and in Innsbruck. In 1874 he took his degree in Vienna. Arlt advised him to get a working knowledge of general surgery before devoting himself to ophthalmology, and consequently he worked for 2 years under Billroth, during which time antisepsis was introduced and Lister himself visited Vienna. From 1876 to 1881 Fuchs was assistant at Arlt's clinic, which laid the basis of his encyclopaedic knowledge of everything pertaining to ophthalmology. During this period he wrote his first large scientific work, about sarcoma of the uvea—a masterly treatise which should be studied by everybody who wants to do research work in the ophthalmological field.



Ernst Fuchs

This work, undertaken to qualify as a *Docent* of the Vienna University, made Fuchs world-famous, and at the age of 30 he was called to the newly created chair of ophthalmology at the University of Liège. He stayed for 4 years and, amongst other scientific publications, he wrote another book which carried his name all over the world. This was a work on the prevention of blindness which, as shown by the table of contents, dealt with eye diseases on a hereditary basis, in childhood, in school age (dealing with the aetiology and prophylaxis of myopia), and as a result of general illness, and contagious eye diseases (particularly blennorhoea and trachoma). It then dealt with the influence of occupation, social environment including illumination, food and cleanliness), and climate. Finally it described the organization of treatment and prophylaxis.

This classic work paved the way for his return to Vienna in 1885, when he became professor there at the age of 34. For nearly 30 years, until 1914, when he retired voluntarily, he remained professor of ophthalmology in Vienna. His influence can hardly be over-estimated. He was generally regarded as the greatest ophthalmologist of his time, and patients came to see him from all over the world. He reformed the teaching in ophthalmology. Every one of his own lectures was a masterpiece, and his school included demonstrations and special courses by his assistants. Foreign doctors flocked to the Vienna clinic, and Fuchs himself gave courses for them in their own language, mostly in English. He had the final say in ophthalmological appointments in Austria and hardly anybody became a lecturer, a professor, or head of an eye hospital, who had not studied under him and had been recommended by him.

During all these activities, Fuchs continued with his scientific work. The number of his publications exceeds 250. Many eye conditions were described by him for the first time, some of which are the following: Blepharochalasis and ptosis myotrophica of the lids; herpes iris of the conjunctiva; episcleritis periodica fugax; heterochromia of the iris; the so-called 'Fuchs coloboma'; retinitis circinata; the black spot in the macula; gyrate atrophy of the choroid; detachment of the choroid after cataract extractions; the diffuse form of choroid sarcoma; and sympathetic ophthalmia following a necrotic sarcoma in the other eye.

No publication, however, contributed so much to the fame of Fuchs as his text-book, first published in 1889. In the introduction he says he wrote this book because he resented it if students took notes during his lectures instead of paying attention to his words. He therefore wanted to provide them with a book where they could find the essence of what he had to tell them and to which they could refer later in life. This book has been called the bible of the ophthalmologist. It has been translated into many languages, amongst them into Chinese and Japanese. Twelve editions came from Fuchs own hand, and altogether 18 editions were published, the last in 1945, 46 years after the first edition and 15 years after Fuchs' death, which took place suddenly from coronary thrombosis at the age of nearly 80, shortly after his return from one of his journeys. His wish for a quick end had been granted.

SOUTH AFRICAN ORTHOPAEDIC ASSOCIATION

CLINICAL MEETING, CORONATION HOSPITAL, JOHANNESBURG*

1. *A Case of Resistant Rickets, presented by Dr. Levin, of Dr. Falcke's Paediatric Unit*

The patient was a child aged 12 years with a history of deformities of the limbs since the age of 18 months. He commenced walking at 2½ years. It was not until he was 9 years old, however, that he was first seen at this hospital. On admission, the appearances were those of vitamin-resistant rickets. The child had frontal bossing, dental caries, enlargement of the epiphyses, and a raised alkaline phosphatase. The blood chemistry was otherwise normal. Later, the blood calcium was normal but the phosphatase was always low. He was put on calciferol and shark oil without much effect.

He has bowing of the legs and is unable to walk without calipers, and then with difficulty. At the time of the present admission he was complaining of abdominal pain and diarrhoea and had marked albuminuria. The intravenous pyelogram was normal as was urea clearance. Renal function was found to be 90% normal.

Dr. Levin asked what could be done to help the child, and Mr. Morris put the question, 'Should one straighten the limbs?'

Mr. Southgate. The deformities are not such that they warrant surgical correction. The real fault lies in his muscular hypotonia. If his muscles were adequate, he could walk in the calipers he now has.

Mr. Lunz. Concurred, in that he considered the limbs sufficiently straight. He felt that attention should be directed to the physiological aspect of the problem.

Mr. Edelstein. This is a typical case of renal rickets, but not a R.R.D. in that in the latter case, there is no renal damage, but merely a failure to utilize vitamin D. This boy, has, however, renal damage, and these are usually hopeless cases. Which type of renal rickets this is, he was not prepared to say, as such differentiation calls for a great deal of biochemical investigation. He would certainly not entertain the idea of osteotomy; this would be attacking the wrong end of the problem.

2. *A Case of Acute Osteomyelitis of Infancy, presented by Dr. Morris, of the Orthopaedic Unit*

The patient presented was admitted a year previously with a discharging sinus of the right hip, and shortening, swelling and local heat. He was treated by traction. Pus was cultured on numerous occasions and *Staph. aureus* was ultimately isolated.

This was a typical case of Tom Smith's arthritis with secondary involvement of the femoral shaft in the infective process.

At operation a sequestrum consisting of practically the whole diaphysis of the femur was removed.

Mr. Morris asked how these cases should be treated primarily and how this particular case should be treated in the future.

* Held on 22 November 1957.

Mr. Southgate. What with so many organisms being resistant to antibiotics today, the hip joint should be aspirated or drained as soon as possible in order to reduce intra-articular tension, and allow typing of the organism.

Mr. Edelstein. This was not a true Tom Smith's arthritis, in that it was a primary osteomyelitis of the femur with secondary destruction of the head of the femur and dislocation at the hip. For the rest, he was in full agreement with Mr. Southgate.

Mr. Craig stated that it was criminal not to drain the hip joint once the diagnosis had been made; in his experience at the Transvaal Memorial Hospital, only in those cases in which this had been done had the head of the femur been saved.

Mr. Lewin mentioned that 3 cases treated at the Coronation Hospital on conservative methods had done well and he felt that this should be given a chance first.

Mr. Moller: In these cases of osteomyelitis, if one happens to strike the right antibiotic one frequently gets a good response, and in such cases one should continue conservatively. One often sees in such cases a reincorporation of the sequestra. If, however, one is not managing to control the infection, one must let the pus out and, if present, sequestra must be removed.

3. *A Case of Multiple Periostitis with Eosinophilia, presented by Dr. Abramowitz, of Mr. Lewin's Surgical Unit*

The patient was a 44-year-old male, a labourer by occupation. He complained of pain in the right leg of 7 years' duration. He dated the onset of pain from some form of trauma, but was not sure whether the tibia had been fractured. There was occasional swelling of the knees. He had a fever of undiagnosed type during the War. There was a history of chest pathology with consolidation at the lung bases, which has since cleared up.

On examination the right tibia was found to be swollen and tender to percussion, with overlying distension of veins.

On X-ray esamination it was considered that the plates showed sclerosis of the cortex in the right tibia, the fibulae and femora. The same changes were to be seen in the radius and ulna on both sides.

On 22 October 1957 a bone biopsy was performed. There was no evidence of plasma cells, but there was a suggestion of areas of necrosis. Pain in the right leg disappeared after the biopsy.

All the bio-chemical tests carried out gave normal results. 24,000 white cells per c.mm. (40% eosinophils). The hydatid complement-fixation test, agglutination tests and marrow investigations were negative. Tests for specific disease gave mildly positive to negative results. The positive result was probably false.

The diagnosis that springs to mind is specific disease—either syphilis or yaws. The raised eosinophil count suggested a possibility of eosinophil granuloma. That this was a case of osteitis deformans was doubtful.

Mr. Edelstein felt that there was no abnormality radiologically in either of the femora or upper extremities, the apparent changes being due to the nature of the films. He suggested the taking of tomographs, because this might well be a case of osteoid osteoma.

Mr. Lewin. The significant feature in this case is the high eosinophilia, which so far cannot be explained.

Mr. Moller. This is a typical case of sabre tibia due to one of three causes; syphilis, Paget's disease or rickets.

Dr. Seimon. Marked eosinophilia may be a feature of syphilis.

Mr. Lewin. Eosinophilia is usually a feature of acute syphilis and in this patient the condition is certainly not acute.

Mr. Southgate. Another condition associated with eosinophilia is Boeck's sarcoidosis.

Mr. Lunz. The most significant thing is that the patient has been rendered symptom-free by the biopsy.

4. *A Case of Cystic Bony Lesions of Carpus, presented for diagnosis by Dr. Seimon, of Mr. Morris' Orthopaedic Unit*

The patient, aged 40 years, complained of pain and swelling of the right wrist of 2 years' duration with increased severity during the last 5 months. The pain was confined to the wrist. In addition, there had been pain in the left wrist during the past 5 months, but this was thought to be due to a ganglion. There was nothing of significance apart from mild arthritis in the past history.

On examination, the right wrist was found to be warm, and movement was markedly restricted. There was no sensory disturbance.

X-ray examination showed numerous cystic areas in the carpus, the base of the second metacarpal, and the end of the radius. Similar small cysts are present in the heads of the humeri.

White cells 21,000 per c.mm. (46% eosinophils). Sedimentation rate 15. Positive hydatid complement-fixation test, but negative skin test. Strongly positive W.R. Examination for ascaris in the stool and for bilharzia gave negative results. Tests for Bence-Jones protein negative. Mantox negative.

Biopsy gave no evidence of parasitic infiltration or tuberculosis, but merely suggests a villous synovitis.

Mr. Morris. The eosinophilia is probably due to syphilis or worms, and the changes in the wrist due to chronic synovitis.

Mr. Edelstein. Clinically and radiologically it looks like tuberculosis. As the Mantoux is negative, the condition is probably Boeck's sarcoidosis.

Mr. Moller said that the consensus of opinion was that this was a case of sarcoidosis, but suggested the possibility of a low-grade chondrosarcoma secondarily invading bone.

5. *A Case of Osteoporosis of the Spine and Swelling of the Leg, presented by Mr. Tanne, of the Surgical Unit*

The patient, aged 46 years, was admitted 1 week ago with pain and swelling of the right lower extremity. In April 1951 she was admitted for an epulis of the upper jaw and bronchiectasis. In February 1952 she had thrombophlebitis of the right leg and gave a history of injury to the back in 1951. It was found that her liver was enlarged and she had blood in the stools. In November 1952 she was again admitted with a fracture of the spine and osteoporosis of unknown origin. W. R. positive. In November 1954 she was again admitted for bronchiectasis and it was found that she had a positive patch test but the sputum was negative. In April 1955 she was admitted with an acute abdomen. X-ray examination revealed that she had osteoporosis of the spine with vertebral collapse.

When admitted a week ago she had pain and swelling of the right leg and thigh. There was no history of trauma, but she gave a history of bleeding from the gums and *per rectum.*

She was found to have spongy pigmented gums and a palpable liver. The central nervous system was normal except that the reflexes in the legs were rather brisker than normal. The right lower extremity was hot, swollen and tender and the inguinal glands were enlarged. There was dense brawny induration of the right calf. She has a tender lumbo-dorsal gibbus.

Investigations: Reversal of albumim/globulin ratio. No Bence-Jones protein. Gross disturbance of liver function. Reversal of neutrophil and lymphocyte ratio. Eosinophilia. Sedimentation rate 50.

Biopsy showed Gland-haemosiderosis and skin-thickening, but no other abnormality.

X-rays: Gross osteoporosis of the spine with collapse of several vertebra. Osteoporosis of the pelvis and bones of the right lower extremity.

Differential Diagnosis. Haemosiderosis: supported by gland biopsy and liver changes. (2) Scurvy: This is the probable diagnosis, being supported by limited joint movement at the knee, thickening of the skin, osteoporosis, and the fact that the original supposed thrombophlebitis was probably in actual fact haemorrhage into the muscles.

Mr. Joffe suggested the performance of a reticulocyte response test.

Mr. Morris. This is a common problem among the Bantu—the nutritional osteoporosis as opposed to senile osteoporosis in the European. They do not respond well to anabolic hormones.

Mr. Lewin. These scorbutic changes often cause severe deformities such as dislocation of the knee.

Mr. Moller. It would appear that the consensus of opinion is that this is a case of scurvy, and in such cases adequate nursing should obviate the onset of deformities.

6. *The Rehabilitation of an Amputee, demonstrated by Dr. Seimon, of Mr. Morris' Orthopaedic Unit*

The patient developed gangrene of a toe in 1952 and was found to have a positive W.R. and sub-periosteal osteitis. His right leg was amputated in 1952 and his left leg and right arm between 1952 and 1956.

The patient walks well with his lower-limb prostheses and has good control of a double-hook-type prosthesis for the right arm. He is now working in the hospital as a lift operator.

7. *X-rays of a case of Neurofibromatosis, shown by Mr. Lewin, of the Surgical Unit*

The patient had died at the age of 53 years. Many years before admission he had experienced pain in the neck with increasing

weakness. There was tenderness over the cervical spine and anaesthesia in the C3 and 4 segments on both sides. Lumbar puncture proved of no assistance in arriving at a diagnosis.

X-ray examination showed spiculated ossified substitution for the spinous processes of C3 and 4.

Laminectomy was performed and a swelling was found in relation to the laminae and spinous processes of C3 and 4. Portion of this swelling was within the lamina and was compressing the dura, while portion was without. The swelling bore no relationship to any visible nerve.

Section of the swelling showed it to be a neurofibroma containing bone. It was of the type that has malignant potential.

The patient's condition did not improve after the removal of the swelling and he has since died. At autopsy it was found that the entire spinal column was infiltrated by a spindle-cell sarcoma of neurological origin.

8. *A Case of Tuberculosis of the Spine with Paralysis of the Legs, presented by Mr. Morris of the Orthopaedic Unit*

The patient was admitted in October 1957, giving a history of pain in first one leg and then the other and later in the back, the onset having been towards the end of 1956. The legs had become progressively weaker.

On examination the ankle jerks were found to be present, but there was a flaccid paralysis of both lower extremities.

X-ray examination showed a typical tuberculous lesion at the D11/12 level with narrowing of joint space and the suggestion of a paravertebral abscess. A myelogram demonstrated a complete block.

Mr. Morris remarked that it was the first case of a purely flaccid tuberculous paralysis that he had seen.

Mr. Craig said he had dealt with a similar case who had presented with complete flaccid paralysis and anaesthesia of both lower extremities of some standing. An antero-lateral decompression was performed, and the patient made a complete recovery. In the light of this experience he suggested that the same course of treatment should be carried out in the case under consideration.

Mr. Tanne. Surely the demonstration of a complete block is sufficient indication for decompression.

Mr. Morris. The block may be due to oedema, and may not be a mechanical block.

Mr. Southgate suggested that conservatism should be tried first and, if not successful, one should then resort to surgery.

Mr. Moller suggested an early fusion to adequately arrest the lesion, with a view to later decompression.

TRANSVAAL SOCIETY OF PATHOLOGISTS

SUMMARIES OF THE SCIENTIFIC PAPERS*

(*a*) HISTOCHEMICAL DEMONSTRATION OF AN ACETYLCHOLINESTERASE IN THE OVA OF SCHISTOSOMA MANSONI

(*b*) ON THE ENZYME HISTOCHEMISTRY OF OSTEOCLASTOMATA

Dr. W. J. Pepler, Institute for Pathology, Pretoria

(*a*) By using acetylthiocholine iodide and o-acetyl-5-bromoindoxyl as substrates in association with various inhibitors it has been possible to demonstrate an acetylcholinesterase in the nervous system of the miracidium of *Schistosoma mansoni*. The material used consisted of frozen sections of the livers of 6 bilharzial infected mice.

It was suggested that the enzyme plays a vital role in the motor activity of this very active parasite and that the use of specific acetylcholinesterase inhibitors might possibly offer a new approach to the control of bilharziasis.

(*b*) In a histochemical study of two osteoclastomata and one giant-cell epulis it was possible to demonstrate a strong acid phosphatase in the giant cells and an alkaline phosphatase localized to the stromal cells and blood-vessels only. Neither of these cell types showed esterase activity.

These findings would then suggest that the giant cells are functionally different from the stromal cells and that the giant cells are still very active and not degenerated cells as has been suggested by some authorities.

THE RELIABILITY OF THE BACITRACIN SCREEN TEST AS A MEANS OF IDENTIFYING HAEMOLYTIC STREPTOCOCCI

Dr. J. A. W. Dressler, S.A.I.M.R.

A preliminary report was given on the investigation of 410 strains of β-haemolytic streptococci. The recommended concentration of 5 units of bacitracin per ml. was used with the disc technique. The first 50 specimens were incubated both aerobically and anaerobically and the succeeding ones planted as surface cultures and incubated aerobically. Of 295 sensitive strains 2·3% were found to be false positives and of 115 resistant strains 4·5% false negatives, as compared with Maxted's 1·7% and 2·5% respectively.

ACUTE REVERSIBLE HEART FAILURE: CORRELATION BETWEEN THE THIAMIN-EXCRETION TEST AND RESPONSE TO TREATMENT WITH THIAMIN IN THE SOUTH AFRICAN BANTU

Dr. W. M. Politzer, S.A.I.M.R.

In order to determine whether thiamin deficiency plays any part in acute reversible congestive cardiac failure, which is a relatively common condition in the South African Bantu, thiamin excretion and saturation tests were carried out and the results obtained were compared with the response of these patients to thiamin treatment. No correlation was found and this confirms the conclusions reached by other workers.

THE SIGNIFICANCE OF THE VI-REACTION IN TRACING TYPHOID CARRIERS

Dr. V. Bokkenheuser, S.A.I.M.R.

By mathematical analysis of published studies it was possible to show that the probability of a person's being a typhoid carrier varied with the Vi-titre. A final titre of 1 : 5 gave a 0·8% probability of being a carrier and 99·2% of being healthy. A titre of 1 : 10 gave a 1·4% probability of being a typhoid carrier and a 98·6% chance of being healthy, while with a titre of 1 : 20 the chances of being a typhoid carrier or healthy were 4·3% and 95·7% respectively. These results suggested that it would be worth while studying the test further, especially it's reproducibility and the criteria used in selecting control groups. When this information is available it might be time to reconsider the value of the test and its role in public-health measures.

* Read at a meeting of the Transvaal Society of Pathologists, Johannesburg, 30 November 1957.

CONGRESS OF S.A. ASSOCIATION OF SURGEONS

The programme has now been issued of the First Congress of the South African Association of Surgeons (a Group of the Medical Association of South Africa) which will be held in the Physiology Lecture Theatre, University of Cape Town Medical School, Observatory, Cape, on Monday, Tuesday and Wednesday, 28-30 April 1958.

Monday morning. Chairman, Mr. G. Sacks, Cape Town.

9.30. Business etc.

9.50. Mr. J. A. Douglas (Johannesburg): 'Technique and results of operative treatment for inguinal herniae in the South Africa Mines'.

11.00. Mr. A. Brown (Cape Town): 'The Differential Diagnosis of Carcinoma of the Large Bowel.

11.30. Mr. W. Trubshaw (Johannesburg): 'The Gastrectomy Problem.

12.00. Dr. G. Efron (Cape Town): 'Analgesia in Pancreatitis with special reference to the effect on Common Bile Duct pressure'.

Monday afternoon. Chairman, Mr. W. Trubshaw, Johannesburg.

2.30. Case Demonstrations, Falconer Lecture Theatre, Groote Schuur Hospital.

4.00. Dr. B. Shandling (Cape Town): 'Acute Osteomyelitis of the neck of the Femur'.

4.30. Mr. J. Hamman (Vereeniging): 'Clinical Patterns in Intestinal Strangulation.

5.00. Mr. D. J. du Plessis (Cape Town): 'The Pathology of Parotid Salivary Gland Obstructions.

Tuesday morning. Chairman, Prof. J. H. Louw, Cape Town.

9.30. Case Demonstrations, Falconer Lecture Theatre, Groote Schuur Hospital.

11.00. Dr. E. Samuel (Johannesburg): 'A new method of Operative Cholangiography'.

11.50. Dr. C. J. Uys (Cape Town): 'The Lymphomata of the Gastro-Intestinal Tract'.

1 p.m.-3.30 p.m. Luncheon in the Medical Residence, Observatory, Cafe. During the latter part of the lunch there will be a Panel Discussion on 'The Management of Haematemesis': Prof. A. Kark (Durban), Mr. W. Trubshaw (Johannesburg), and Prof. J. H. Louw (Cape Town): Chairman, Mr. A. Radford (Durban).

Tuesday afternoon. Chairman, Mr. J. A. Douglas, Johannesburg.

4.00. Prof. A. Kark (Durban): 'The Pathology of non-Malignant Strictures of the Colon and Rectum'.

4.40 Mr. T. Schrire (Cape Town): 'The Aetiology of Facial Cancer'.

5.10. Mr. M. Arnold (Johannesburg): 'Regional Anaesthesia in Surgery'.

Wednesday morning. Chairman, Mr. A. Colley, Port Elizabeth.

9.30. Mr. K. Colsen (Johannesburg): 'Aspects of Cervical Spine Injury'.

10.00. Prof. J. H. Louw (Cape Town): 'Observations on Pathogenesis and Treatment of Intestinal Atresia'.

11.00. Mr. D. Chapman (Durban): 'Diseases of the Penis, with special reference to Cancer'.

11.30. Panel Discussion on 'The Management of the Jaundiced Patient': Mr. J. A. Douglas (Johannesburg), Mr. A. L. McGregor (Johannesburg) and Mr. G. Sacks (Cape Town).

Wednesday afternoon. Chairman, Mr. Sweetapple, Durban.

2.30. Mr. A. Walt (Cape Town): 'Carcinoma of the Thyroid'.

3.00. Prof. F. van Zyl (Cape Town): 'Views on the Treatment of Carcinoma of the Breast'.

4.10. Mr. E. Barker (Durban): 'Unusual presentation of Peripheral Vascular Disease affecting the Upper Limbs in Young Women'.

Social Events

Besides the luncheon on Tuesday (see above), a Dinner will be held on Monday evening at the Vineyard Hotel, Newlands, for the surgeons and their wives. Formal dress.

On Wednesday at 6 p.m. Mr. J. Duminy, Principal and Vice-Chancellor of the University of Cape Town, will entertain the surgeons and their wives to a Cocktail Party.

Tea will be provided in the Students' Tea Room every day at 10.30 a.m. and 3.30 p.m.

Questionnaire No. 2

All surgeons intending to be present are asked to complete Questionnaire No. 2 (irrespective of whether the first one was sent in) and to return it to Mr. D. J. du Plessis, Department of Surgery, Medical School, Falmouth Road, Observatory, Cape.

PNEUMOCONIOSIS BOARD: CONSTANT ATTENDANT ALLOWANCE

Under the Pneumoconiosis Act a miner may be awarded an allowance in respect of a constant attendant, subject to certification by a medical practitioner that his condition makes a constant attendant necessary. For the information of medical practitioners the following memorandum on the subject (MD 389) has been received by the Pneumoconiosis Compensation Commissioner, and will be printed on the reverse side of the certificate form, which is available either in Afrikaans or English.

CONSTANT ATTENDANT ALLOWANCE

1. The Pneumoconiosis Board is empowered by Section 76 of the Pneumoconiosis Act (No. 57 of 1956), to award an allowance in respect of a constant attendant. The section reads as follows:

(1) When the condition of a miner to whom a pension has been awarded under any provision of this Act is such that, in the opinion of the board, he needs a constant attendant, the board may on application by such miner award him a monthly allowance not exceeding the sum of seven pounds and ten shillings, subject to the submission by him every three months, or at such other intervals as the board may determine, of a certificate issued by a medical practitioner to the effect that the condition of the miner is such that he needs a constant attendant.

(2) Any such allowance may at any time be withdrawn or reduced as the board deems fit.

2. It is clear from the foregoing that—

(i) the award of the allowance is in the discretion of the board; it is not part of the miner's pension and he is also not entitled to it as a right;

(ii) the award is subject to a supporting certificate issued by a medical practitioner;

(iii) the physical impairment of the applicant may be due to causes other than tuberculosis or the impairment of his cardio-respiratory organs by pneumoconiosis; and

(iv) the attendant need not be a nurse or trained person.

3. The Act defines 'tuberculosis' and 'pneumoconiosis.' As regards the last-mentioned disease four stages are defined as follows:

'pneumoconiosis' means disease of the cardio-respiratory organ (by whatever means discovered) which has been caused by exposure to dust in the course of work in a dusty atmosphere; and a person shall be deemed to be or to have been suffering from pneumoconiosis—

(*a*) in the first stage, if his cardio-respiratory organs have been found to be or to have been permanently affected by pneumoconiosis, whether or not his capacity for manual work is or was permanently impaired thereby;

(*b*) in the second stage, if his cardio-respiratory organs have been found to be or to have been permanently affected by pneumoconiosis as a result of which he is or was permanently incapacitated from performing work more strenuous than moderate manual work;

(*c*) in the third stage, if his cardio-respiratory organs have been found to be or to have been permanently affected by pneumoconiosis as a result of which he is or was permanently incapacitated from performing work more strenuous than light manual work;

(*d*) in the fourth stage, if his cardio-respiratory organs have been found to be or to have been permanently affected by pneumoconiosis as a result of which he is or was permanently incapacitated from performing any manual work.

4. Although according to the foregoing definition, the incapacity caused by the first three stages of pneumoconiosis may still leave a person theorectically fit for some kind of work, there may be present some other incapacity which renders him unable to attend to his daily needs, e.g. severe arthritis accompanying the pneumoconiosis. Conversely the fourth stage of pneumoconiosis does not necessarily imply that a person is unable to attend to his daily needs and there may be cases where, although a person is unfit for work he is still capable of looking after his daily requirements.

5. The condition of the miner may be complicated by a finding of tuberculosis with or without pneumoconiosis. The degree of care and attention which such a person requires is variable and only the medical practitioner attending him regularly will be able to certify on this point.

6. Nobody realises better than the Board the danger of reading too much or too little into a statutory provision such as that

quoted above. Apart from the obvious case where a person is so ill and frail as to be helpless or incapacitated by physical or mental defects (e.g. blindness, loss of limbs, etc.) it is often difficult to judge whether a constant attendant is needed.

The Board is not a medical body and it has no desire to prescribe or dictate views on a matter which involves the submission of a certificate by a medical practitioner. Since, however, the final decision rests with the Board it might be helpful to indicate to the medical profession that in 'difficult' cases questions such as the following afford some guidance:—

(i) Is the applicant subject to some form of attack, whether occurring at regular or irregular intervals, which makes it unsafe for him to be left by himself for any length of time?

(ii) Although failing in health, is the applicant capable of attending to his daily needs such as dressing, bathing, shaving and feeding or does he require assistance for these needs?

(iii) Is the condition permanent or will it endure for such length of time that a constant attendant is warranted?

(iv) Although not suffering from any severe illness, is the applicant unable to attend to his physical needs and is he not able to be left alone on account of old age (senility)?

(v) Is the applicant unable to help himself on account of loss of limb, blindness or incapacitation by osteoarthritis or some other disease?

(vi) Is the mental condition of the applicant such that, although not certifiable, he cannot be left to attend to himself?

(vii) Is the applicant partially or completely paralysed as a result of a stroke and consequently unable to dress, feed himself, etc?

(viii) Is the applicant, although not suffering from any defined illness, confined to bed as a result of general bodily weakness and debility and not able to attend to his needs?

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

VACANCY—ASSISTANT EDITOR

Applications are invited from medical practitioners for the post of Assistant Editor in the service of the Medical Association of South Africa at its Head Office in Cape Town.

The salary scale attaching to the post is £1,250×50—1,750 per annum, plus an annual cost-of-living allowance of £176 for single men and £352 for married men. The commencing salary will be determined according to experience.

The successful applicant must contribute to the Association's Superannuation Fund. He will also be expected to assume duty as soon as possible after appointment.

Applications must reach the Secretary, Medical Association of South Africa, P.O. Box 643, Cape Town, before 31 March 1958.

A. H. Tonkin
Secretary

Medical House
Cape Town
24 February 1958

VAKATURE—ASSISTENT-REDAKTEUR

Aansoeke word van geneeshere ingewag vir die betrekking van Assistent-Redakteur in diens van die Mediese Vereniging van Suid-Afrika, by die Hoofkantoor te Kaapstad.

Die salarisskaal aan die pos verbonde is £1,250×50—1,750 per jaar, plus 'n jaarlikse duurtetoeslag van £176 vir 'n ongetroude en £352 vir 'n getroude man. Die aanvangssalaris sal volgens ondervinding bepaal word.

Die suksesvolle kandidaat moet by die Vereniging se pensioenskema aansluit. Hy sal ook verwag word om so spoedig moontlik na aanstelling diens te aanvaar.

Aansoeke moet die Sekretaris, Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, bereik vóór 31 Maart 1958.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
24 Februarie 1958

NEW PREPARATIONS AND APPLIANCES: NUWE PREPARATE EN TOESTELLE

AMBU RESUSCITATOR
AMBU FOOT SUCTION PUMP

Westdene Products (Pty.) Ltd. announce the arrival of these two life-saving aids manufactured by the Testa-Laboratorium, Copenhagen, and supply the following information:

Both of these new pieces of equipment are ready for use anywhere and at any time; they do not require oxygen cylinders or water pressure. For this reason they save valuable time in emergency.

The Ambu Resuscitator is small, compact and light. It can be used at the moment of emergency without need of connections or setting of gauges. It is worked by fingertip control and can be operated for hours without tiring the operator. The bag provides 400-1300 ml. of air with one compression and *maintains a physiologically correct rhythm* through its self-inflating construction. A special valve is fitted to the Ambu Resuscitator which provides for oxygen-enriched mixtures if so desired.

The Ambu Suction Pump, foot-operated, provides negative pressure of 280-300 mm. of mercury. It is light in weight and operates without electric motors or water pressure and will continue operating with a full bottle or without any bottle at all.

The outstanding advantages of the two units are simplicity of operation, rugged construction, and ability to work *wherever* respiratory failures occur.

Sole South African importers and distributors: Westdene Products (Pty.) Ltd., P.O. Box 7710, Johannesburg.

PASSING EVENTS : IN DIE VERBYGAAN

The 4th World Assembly of the Israel Medical Association will be held in Israel on 12-24 August 1958. The Assembly will coincide with the Festival marking the 10th anniversary of the foundation of the State of Israel. The meetings will start in Tel Aviv and will be continued in Haifa and Jerusalem. The scientific sessions will deal with the medical integration of immigration mainly covering the diseases specific to Israel as a land of mass immigration. Symposiums and panel discussions will be held bearing on health problems connected with immigration. In addition there will be discussions for specialist groups in various branches. Visits will be arranged to hospitals and clinics to give the opportunity of seeing and understanding the medical problems of these institutions. A carefully prepared intinerary has been drawn up to enable participants to visit the whole country, including historical sites, settlements and branches of industry and agriculture. Peltours, P.O. Box 394, Tel Aviv, have been appointed as official travel agents. The Congress office is at Beth-Harofe, 1 Heftman Street, Tel Aviv, and information may be obtained in South Africa from Dr. Cyril Adler, 701 Ingram's Corner, Twist Street, Hospital Hill, Johannesburg.

* * *

Deaths associated with Anaesthesia and Surgery. The Department of Anaesthesia, University of Pretoria, has since July 1956 been doing research for the C.S.I.R. on the causes of deaths associated with anaesthesia and surgery. A paper by Dr. O. V. S. Kok, Chief Anaesthetist, Pretoria General Hospital, which was presented at the South African Medical Congress, Durban, in September 1957 and published in this *Journal* on 15 February 1958 (p. 180), recorded results of this research to date, based on data received from certain

hospitals. According to this report it appears that the complication of cardiac arrest is steadily increasing. Out of 200 deaths associated with the administration of an anaesthetic, 132 were cases of cardiac arrest occurring on the operating table. It is intended to make further research into this complication, including cases in which the patient eventually recovered.

Dr. Kok has now circulated a letter to all hospitals in South Africa (including Native mine hospitals) asking for systematic returns of information on printed forms at 3-monthly intervals, concerning every death associated with anaesthesia or operation. Further details and specimen forms are obtainable from Dr. O. V. S. Kok, Department of Anaesthesia, Pretoria General Hospital, P.O. Box 667, Pretoria.

* * *

The Second World Congress of the International Federation of Gynaecology and Obstetrics, which will be held in the Queen Elizabeth Hotel, Montreal, Canada, on 22-28 June 1958, will have on its programme eight main lectures given by eminent scientists whose studies and research are related to the field of gynaecology and obstetrics. The guest speakers and the title of their lectures are as follows: Prof. Murray L. Barr, Department of Microscopic Anatomy, University of Western Ontario, London, Canada: '*Tests of chromosomal sex and their application to clinical problems*'. Prof. Hermann Bautzmann, Anatomisches Institut, Hamburg, Germany: '*Comparative Studies on the Histology and Function of the Animal and Human Amnion*'. Prof. Roberto Caldeyro-Barcia, Chief of the Section of Obstetrical Physiology, Faculty of Medicine, Montevideo, Uruguay: '*Contractility of the Human Gravid Uterus and its application to the Obstetrical Clinic*'. Prof. G. W. Harris, Head of the Department of Neuroendocrinology, Maudsley Hospital, London, England: '*Relationship of the Central Nervous System to Pituitary and Reproductive Activity*'. Prof. Charles Oberling, Institute de Recherches sur le Cancer Gustave Roussy, Villejuif (Seine), France: '*The Cytology of the Cancerous Cell*'. Prof. Bradley M. Patten, Chairman, Department of Anatomy, University of Michigan Medical School, Ann Arbor, Michigan: '*The Establishing of Foetal-Maternal Vascular Relations*', Prof. Hans Selye, Director of the Institute on Experimental Medicine and Surgery, University of Montreal, Montreal, Canada: '*Stress in Gynaecology*'. A representative of the USSR (on a subject of his choice). All correspondence should be directed to the Montreal Committee, Second World Congress, International Federation of Gynaecology and Obstetrics, 1414 Drummond Street, Suite 220, Montreal 25, Canada.

* * *

Union of South Africa, Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 21 February, 1958, to 27 February, 1958.

Plague, Smallpox: Nil.

Typhus Fever, Cape Province: One (1) Native case in the municipal area of Steynsburg and one (1) Native case in the Glen Grey District.

Correction: Three (3) Native cases which occurred in the Glen Grey District and which should have been included in No. 49 of 1957, were not reported. One (1) of the three Native cases in the Ngeleni District, which were reported in No. 49 of 1957, has since died.

Poliomyelitis

	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal ..	9	1	—	—	10
Cape Province ..	1	1	4	—	6
Orange Free State..	2	4	—	—	6
Natal	1	—	—	—	1
Totals	13	6	4	—	23

Correction: One (1) European case in Natal which was reported in No. 52 of 1957 has since been diagnosed as not-Poliomyelitis.

Local Authorities		*Eur.*	*Non-Eur.*
Transvaal:			
Germiston Municipality ..	U	1	
Groot Marico District ..	R	1	
Johannesburg Municipality ..	U	3	
Koster District	R	1	
Krugersdorp Municipality ..	U		1
Potgietersrust District ..	R	1	
Pretoria Municipality	U	2	
Cape Province:			
Barkly West Municipality ..	U	1	
Cape Divisional Council ..	U		3
Kenhardt Divisional Council ..	R		1
Mt. Ayliff Village Management Board	U		1
Stellenbosch Divisional Council	R	1	
Orange Free State:			
Bloemfontein Municipality ..	U		1
Kroonstad Municipality ..	U	1	
Paul Roux District	R		1
Smithfield District	R		1
Theunissen District	R		1
Natal:			
Kingsborough Borough ..	U	1	

Correction: One (1) European case in Natal which was reported in No. 52 of 1957 has since been diagnosed as not-Poliomyelitis.

U—Urban R—Rural

CORRESPONDENCE : BRIEWERUBRIEK

ELI LILLY MEDICAL RESEARCH FELLOWSHIP (SOUTH AFRICA)

To the Editor: Applications for the 1958 award of this Fellowship may now be submitted and must reach the undersigned not later than 30 April 1958.

The detailed conditions governing the award of this Fellowship appear in the issue of *Medical Proceedings* dated 8 March 1958 at p. 128. They may also be obtained from the undersigned.

H. A. Shapiro, Ph.D., M.B., Ch.B., F.R.S.S.Af.
Honorary Chairman, Selection Committee, Eli Lilly Medical Research Fellowship, (South Africa)

P.O. Box 1010,
Johannesburg.
28 February 1958

SOUTH AFRICAN SOCIETY OF PHYSIOTHERAPISTS

To the Editor: My attention has been drawn to an incorrect translation into Afrikaans of our letter-heads. The English, 'South African Society of Physiotherapists' should be, in Afrikaans, 'Suid Afrikaanse Fisioterapie Vereniging', and not 'Fisiogeneeskundige Vereniging.'

As some of these incorrect letterheads have been received by doctors, and as some of our members have copied our translation for their own note-paper or cards, I should like, through your *Journal*, to express our sincere regret for any offence caused and advise that steps have been taken to correct the error. Our members have been advised accordingly.

Your cooperation in publishing this note will be much appreciated. I am anxious to dispel any misconceptions that this error might create.

G. S. Schermbrucker
Chairman, Western Province Branch S.A. Society of Physiotherapists
Geneva House
Cape Town
3 March 1958